Insomnia, Dysfunctional Beliefs About Sleep, Hopelessness and Depression Among Older Adults: The Development and Testing of a Path Model

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

STATEMENT OF AUTHORSHIP

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Abstract

Insomnia increases the risk of developing depression among older adults. No study, however, has attempted to explain how insomnia predicts depression from a cognitive perspective. The primary aim of the current study was to test a path model that explored whether dysfunctional beliefs about sleep and hopelessness explain how insomnia influences depression among older adults. It was hypothesised that insomnia would predict depression, both directly and indirectly, via dysfunctional beliefs about sleep and hopelessness. A second exploratory aim tested whether the model differed according to whether or not participants were likely to have a physiological sleep disorder. A community sample of 218 older adults aged from 65 to 96 years, completed the Insomnia Severity Index, Dysfunctional Beliefs and Attitudes About Sleep 10-Item Scale, Beck Hopelessness Scale, Snoring Tiredness Observed Pressure Scale, and Restless Legs Syndrome Questionnaire. Out of the 218 participants, 171 completed the Centre for Epidemiologic Studies Depression Scale three months later. The model was tested using a longitudinal path analysis design. Initial results indicated that the overall model fit was poor. One modification that involved the addition of a direct path from insomnia to hopelessness resulted in an excellent model fit. Results further demonstrated that the revised path model was non-invariant between older adults who were likely to have a physiological sleep disorder and older adults who were unlikely to have a physiological sleep disorder. It was concluded that dysfunctional beliefs about sleep and hopelessness partly explain how insomnia influences depression among older adults, irrespective of the likelihood of having a physiological sleep disorder.

The older adult population, defined as individuals aged over 65 years (Thane, 1989), is growing at a rapid rate worldwide. 'Population ageing' is characterised by an upward shift in the age structure, where the proportion of younger people declines as the proportion of older people increases (World Health Organisation [WHO], 2006). This shift in the global population structure is the result of sustained low levels of fertility combined with increasing life expectancy (WHO, 2006). The WHO reported that the proportion of older adults throughout the world is forecasted to triple over the next 40 years. For instance, there were 600 million older adults constituting approximately 10% of the total global population in the year 2000 (WHO, 2006). It is predicted that the number of older adults will increase to 2 billion by 2050, which will represent approximately 30% of the total global population (WHO, 2006). This international ageing population trend is also occurring within Australia.

The proportion of older adults in Australia has grown steadily during the 20th century and it is projected to grow further during the 21st century (Australian Bureau of Statistics [ABS], 2009). In 1901, there were 150,000 people aged 65 years and over living in Australia, comprising 4% of the total Australian population (ABS, 2009). By 2007, this number increased to approximately 2.8 million or 13% of the total population. The ABS predict that by 2056 the older population will have grown to between 6 million and 6.3 million or around 25% of the projected total population of Australia (ABS, 2009). As a result of this trend, the Australian Institute of Health and Welfare (AIHW, 2009) has declared research into Australian older adults a national priority, in order to continue improving the knowledge of and services for this growing population.

The consequences of an ageing population are likely to be felt in almost every sector of society (Kovner, Mezey, & Harrington, 2002). One particular area of the community that will feel this demographic change most profoundly is the healthcare sector (Crimmins, 2004). In Crimmins' (2004) review, it was reported that older adults experience more physical health

problems, including sensory impairments, cognitive deficits, chronic medical conditions, and sleep difficulties, compared to younger adults. In addition, older adults are requiring increased assistance from healthcare professionals such as general practitioners, psychologists, and emergency services (Aminzadeh, Dalziel, & Molnar, 2002; Qualls, Segal, Norman, Niederehe, & Gallagher-Thompson, 2002). With an ageing population, the proportion of older adults experiencing health problems will increase and place greater demand on the healthcare system. Consequently, the field of health has to prepare for a dramatic increase in older adult healthcare needs. This highlights the importance of future research to promote positive ageing and prevent the onset of illness.

Good physical and mental health are essential elements for older adults to remain independent and participate in family and community life (Weir, Meisner, & Baker, 2010). Research has indicated that factors such as regular physical activity (Everard, Lach, Fisher, & Baum, 2000), social support (Seeman, Lusignolo, Albert, Berkman, 2001), community involvement (Greenfield & Marks, 2004), and adaptive/flexible thinking (von Faber et al., 2001) are positively associated with healthy ageing. Despite common challenges that can surface during the ageing process (e.g., changes in sleep, sensory impairments, slowed mobility), most older adults are able to cope with age-related stressors (Weir et al., 2010). A significant proportion of older adults, however, find it difficult to adapt to these changes during the ageing process. One of the major changes that commonly accompanies the ageing process is the disruption of an individual's daily sleep-wake cycle.

Sleep and Ageing

Sleep complaints are common in all age groups (Feinsilver, 2003; Ford & Cooper-Patrick, 2001). Older adults, however, are particularly vulnerable to sleep problems (Ancoli-Israel & Ayalon, 2006). In an historic epidemiological sleep study, over 9000 community

dwelling older adults completed self-report measures on sleep disturbance (Foley, et al., 1995). Foley and colleagues found that disturbed sleep was common among older individuals, reporting that 88% complained of occasional sleep problems. More specifically, 42% reported ongoing difficulties with initiating or maintaining sleep and 52% reported having inadequate sleep that interfered with their daytime alertness. More recently, a comprehensive review by Ohayon (2002) suggested that 15% to 52% of community older adults reported difficulties initiating sleep, 20% to 65% reported difficulties maintaining sleep, 15% to 54% reported early morning awakenings, and approximately 12% reported non-restorative sleep. Collectively, these results demonstrate that many older adults report persistent sleep problems.

Sleep accounts for approximately one third of human life (Dement & Mitler, 1993). It is a vital physiological process with important restorative functions (Klerman & Dijk, 2008; Wolkove, Elkholy, Baltzan, & Palayew, 2007). Rather than being one uniform state, sleep has two distinct phases: rapid eye movement (REM) sleep and non rapid eye movement (non-REM) sleep (Feinsilver, 2003). Non-REM sleep contains four gradually merging stages, which commences from lighter sleep (stage one and two) and proceeds to deeper sleep (stage three and four). Sleep is staged by monitoring brain waves via electroencephalogram, muscle activity via electromyography, and eye movements via electrooculography. REM sleep consists of relatively low voltage, mixed frequency brain wave activity somewhat similar to stage one non-REM sleep or wakefulness (Feinsilver, 2003). Feinsilver reported that one of the prime characteristics of REM sleep is a very low level of muscle tone. Since REM sleep combines increased brain activity with decreased muscle tone, researchers have reported that this is when dreaming most likely occurs (Feinsilver, 2003). Most individuals generally pass through the stages of non-REM sleep with an REM period every 90 to 120 minutes during the night (Shochat, Pillar, & Malhotra, 2007). More stage three and four sleep is generally

observed in the first half of the sleep period, and more REM sleep during the second half (Feinsilver, 2003; Shochat et al., 2007). Although the amount of time an individual needs to sleep can vary greatly, there are some common changes that occur with sleep as people age.

The most striking change in sleep among older adults is the repeated and frequent interruption of sleep by periods of wakefulness (Vitiello, 2006). Older adults are more easily aroused at night by environmental stimuli, suggesting that they may be more sensitive to noises or movements (Zepelin, McDonald, & Zammit, 1984). This is possibly the result of an age-dependent intrinsic lightening of sleep homeostatic processes (Dijk, Duffy, & Czeisler, 2001). Another change in sleep among older adults is a decline in sleep efficiency (the ratio of time asleep to time spent in bed). Shochat et al. (2007) stated that this is most likely the result of longer delayed sleep onset (sleep latency) and more awakenings during the night compared to younger adults. In addition, older adults experience less REM and stage four non-REM sleep than younger adults (Phillips & Ancoli-Israel, 2001; Shochat et al., 2007). For example, the relative amount of REM sleep declines from 40% of sleep time in early childhood to 20% by age 70 (Phillips & Ancoli-Israel, 2001). These changes are indicative of impaired sleep maintenance and depth among older adults, which often contributes to lighter, more fragile sleep, than that of younger adults (Vitiello, 2006). These age-associated increases of night time wakefulness are reflected by increases in daytime fatigue, excessive daytime sleepiness, and increased likelihood of napping during the day (Campbell, Murphy, & Stauble, 2005; Monk, Buysse, Carrier, Billy, & Rose, 2001). These changes suggest an age-related disturbance to the normal circadian sleep-wake cycle.

In addition to the changes in sleep quality across the human life span, the timing of that sleep also changes with aging. The sleep-wake cycle that occurs over a 24-hour period is an example of one's circadian rhythm (Vitiello, 2006). Circadian rhythms are biological rhythms that control many physiological functions. The sleep-wake cycle is synchronised by

the internal core body temperature, endogenous melatonin cycle, and the external light-dark rhythm, which asserts its effect on the sleep-wake cycle through the retino-hypothalamic visual pathway (Ancoli-Israel & Ayalon, 2006). With age, the sleep-wake circadian rhythm becomes less synchronised, resulting in less consistent periods of sleep across the 24 hour period (Ancoli-Israel, 2000).

There is a considerable disparity in the literature concerning circadian rhythms and ageing. The impact of aging on circadian rhythms has recently been comprehensively reviewed by Monk (2005). Monk suggests that with ageing, the circadian amplitude is reduced; there is a circadian phase advance (e.g., the circadian rhythm moves earlier relative to the environment); there is a shortening of the circadian cycle; and the ability to tolerate rapid phase changes (e.g., jet lag) declines. According to Vitiello (2006), however, the available evidence supports only two of Monk's four circadian rhythm assumptions. First, there is a phase advance in the normal circadian sleep cycle, meaning that older adults tend to go to bed earlier, and wake up earlier than younger adults (Duffy et al., 2002; Wolkove et al., 2007). Second, older people have more trouble than younger adults adjusting to the rapid phase shifts of shift work and jet lag, at least in terms of sleep quality, subjective sleep complaints, and performance measures (Bonnefond et al., 2006). As a result, there is partial support in the literature that changes in circadian rhythms influence sleep with advancing age.

Currently, it is not clear whether older people need less sleep, but it appears that nocturnal sleep time declines with age (Feinsilver, 2003; Phillips & Ancoli-Israel, 2001). For instance, infants tend to sleep for 16-20 hours, young children for 10-14 hours, adolescents for 8-12 hours, adults for 7-9 hours, and older adults for 6-8 hours (Phillips & Ancoli-Israel, 2001). Furthermore, Cauter, Leproult, and Plat (2000) found that total sleep time decreased on average by 27 minutes per decade from mid-life until eighth decade. Overall, it has been proposed that the total amount of time spent asleep at night tends to decrease with age.

A recommended healthy sleep pattern for older adults includes a sleep latency less than 30 minutes, 5-10 hours of sleep per night, a sleep efficiency of 85%, and no reported no daytime impairment because of sleepiness (MacFarlane, 2002; Petit et al., 2003). As individuals differ significantly regarding the optimum amount of sleep they need, sleep requirements should be dependent on the sleep levels that prevent an individual's daytime dysfunction or mood impairments, rather than on sleep time averages (Roth, 2007).

In summary, the older adult population is rapidly growing worldwide and will place greater pressure on the healthcare system in the future. A number of changes occur during the ageing process that affect older adults. One of the most profound changes that older adults experience is with their sleep pattern. For instance, older adults wake more often during the night compared to younger adults. Although age-related sleep changes are often a normal part of ageing, many older adults encounter significant sleep difficulties that develop into one or more serious sleep problems.

Insomnia

Insomnia is considered a public health epidemic (Smith, Huang, & Manber, 2005). Insomnia is the most prevalent sleep problem and increases significantly with age (Doghramji, 2006; Foley et al., 1999; McCurry, Logsdon, Teri, & Vitiello, 2007; Neubauer, 2005; Roth, 2007). For example, research has shown that 20-30% of older adults suffer with persistent insomnia compared with 10-15% of adults aged less than 65 years (Doghramji, 2006). Moreover, insomnia is one of the most commonly reported problems by older adults to general practitioners, yet research suggests that insomnia remains one of the most under-recognised, under-treated, and misunderstood health conditions among the elderly (Benca, 2005; Bootzin & Epstein, 2011; Doghramji, 2006; Morin, 2004). With an ageing population,

these findings demonstrate that insomnia presents a major current and future concern for the community.

Insomnia has been classified on the basis of etiology into primary and secondary subtypes (Doghramji, 2006; Neubauer, 2005). Most studies that investigate insomnia refer to the sleep disorder known as primary insomnia. To be diagnosed with primary insomnia, several strict criteria must be met. The first diagnostic criterion of primary insomnia, according to the *Diagnostic and Statistical Manual of Mental Disorders,* 4th Edition, Text Revision *(DSM-IV-TR*), states that primary insomnia is an ongoing difficulty with initiating or maintaining sleep which has been occurring on a regular basis for at least one month (American Psychiatric Association, [APA], 2000). Primary insomnia can be short-term/acute (less than one month), or chronic/persistent (more than one month). The second criterion is that the sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (APA, 2000). The third criterion is that the sleep disturbance does not occur exclusively during the course of a physiological sleep disorder (e.g., obstructive sleep apnoea). The fourth criterion is that the disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder). Finally, the fifth criterion is that the disturbance is not due to the direct physiological effects of a medical condition or substance (e.g., drug abuse, medication).

Most insomnia-related studies (e.g., Morin, 2004) focus exclusively on individuals who meet the diagnostic criteria for primary insomnia. These studies usually investigate healthy individuals who have a specific problem with initiating or maintaining sleep. Although this is an important area of research, many older adults who experience ongoing problems with insomnia are not represented in these studies because they experience comorbid problems such as medical/psychiatric conditions or physiological sleep disorders

(McCurry et al., 2007; Smith et al., 2005; Taylor et al., 2007). For example, in an influential study by Morin and colleagues (1999) that compared behavioural and pharmacological treatment for late-life primary insomnia, 85 participants were excluded from its original sample of 163, which left 78 participants to be randomised to the treatment groups. More recently, a similar designed study conducted by Edinger, Wohlgemuth, Radtke, Marsh, and Quillian (2008) screened 192 participants that presented with sleep problems. Of these, 117 participants did not meet the specific criteria for primary insomnia, leaving a final sample size of 75 participants. In these studies, participants were excluded due to having comorbid physiological sleep disorders; medical or psychiatric conditions; taking prescribed medication; or did not meet the diagnosis of primary insomnia. These are two studies of many (e.g., Edinger & Means, 2005; Sivertsen et al., 2006; Wang, Wang, & Tsai, 2005) that have excluded a significant proportion of their sample based on the *DSM-IV-TR* primary insomnia diagnostic criteria. This has presented an important limitation within the literature because many older adults that have been excluded from this body of research still experience chronic insomnia problems that impair their daytime functioning (McCrae & Lichstein, 2001; Smith et al., 2005; Stepanski & Rybarczyk, 2006; Taylor et al., 2007).

Studies have begun to explore different classifications of insomnia to capture a greater proportion of the community who experience ongoing difficulties with initiating or maintaining sleep. Secondary insomnia, in contrast to primary insomnia, has been defined historically as insomnia resulting from other medical or psychiatric illnesses, medication use, or other physiological sleep disorders (Ancoli-Israel, 2000; Lichstein, Wilson, & Johnson, 2000). Research has reported that secondary insomnia is much more common in the older population than primary insomnia due to an array of often co-occurring problems among this age group (Stepanski & Rybarczyk, 2006), such as medical conditions (Taylor et al., 2007) or physiological sleep conditions (Edinger, 2003; Smith et al., 2005). Lichstein et al. (2000)

further propose that for insomnia to be classified as secondary, fluctuation in its severity should parallel the course of the primary condition. In clinical practice, however, this distinction is often difficult, and sometimes impossible to make, because the onset and severity of chronic insomnia is impacted by interrelated medical, psychiatric, behavioural, and psychosocial factors (Harvey, 2001). Consequently, recent studies have indicated that the term 'comorbid insomnia' be used in reference to insomnia that co-occurs with another medical or psychiatric condition, unless causality has been clearly established (Smith et al., 2005; Stepanski & Rybarczyk, 2006; Taylor et al., 2007).

For the purposes of the current study, insomnia will refer to an ongoing complaint of disturbed sleep, manifested as difficulties in sleep initiation, sleep maintenance, or early morning awakening that cause impairment to daytime functioning (Bastien, Vallieres, & Morin, 2001; Doghramji, 2006; Harvey, 2001). This definition will be used to describe the severity of an older adult's level of insomnia, with a focus on inclusion rather than exclusion to capture a realistic representation of the older adult community.

In summary, insomnia is one of the most common health complaints reported by older adults. Older adults are twice as likely to experience problems with insomnia compared to younger adults. Most studies that investigate primary insomnia focus on obtaining a clinical population that often excludes over half of their original sample, which limits the ability to generalise results to the broader older adult community. Consequently, future research needs to consider investigating a community sample of older adults because many older individuals are excluded from the literature despite experiencing ongoing significant insomnia problems.

Causes of Insomnia

There are many factors that can potentially contribute to the development of insomnia (Bootzin & Epstein, 2011; Glovinsky & Spielman, 2007). Understanding the causes of

insomnia is a particularly complex process among older individuals because there are usually several factors that influence persistent insomnia in this population (Bootzin & Epstein, 2011; Foley, Ancoli-Israel, Britz, & Walsh, 2004; Glovinsky & Spielman, 2007; Quan et al., 2005; Stepanski, Rybarczyk, Lopez, & Stevens, 2003; Vitiello, Moe, & Prinz, 2002). Glovinsky and Spielman (2007) provided a comprehensive insight into the potential causes of insomnia by applying the predisposing, precipitating, and perpetuating framework.

Predisposing characteristics are operative before an episode of insomnia occurs (Glovinsky & Spielman, 2007). These characteristics make an individual more susceptible to developing insomnia. Many predisposing factors appear to be present on an inherited basis (Glovinsky & Spielman, 2007). For example, some people describe being a poor sleeper from infancy and report that poor sleep is consistent throughout their family. This may involve a genetic predisposition to sleep-wake circadian rhythm problems (Monk, 2005). In addition, individuals who tend to have anxious or depressive personality traits often require fewer external stressors to disturb their sleep pattern due to physiological or cognitive hyperarousal (Glovinsky & Spielman, 2007). Furthermore, individuals may describe either being more of an ‘early person’ or ‘evening person’, indicating that they prefer to go to bed early and wake up early, or go to bed late and wake up late. Either sleep preference could become problematic for some individuals when changes in circadian sleep patterns occur later in life (Ancoli-Israel & Ayalon, 2006; Glovinsky & Spielman, 2007; Monk, 2005).

Predisposing factors can also be an acquired characteristic that indirectly may lead to problems with insomnia (Glovinsky & Spielman, 2007). For instance, an individual who experiences a chronic medical condition or chronic pain could lower their threshold to developing insomnia years later (Dijk et al., 2001; Vitiello, 2006). Whether insomnia is inherited or acquired, predisposing characteristics are typically overlooked because they predate the onset of the current insomnia problem (Glovinsky & Spielman, 2007).

Precipitating events are usually easier to identify than predisposing characteristics because they involve changes in an individual's routine that can disrupt both day and night functioning (Glovinsky & Spielman, 2007). Precipitating events are usually readily identifiable at the time disturbed sleep becomes acute. These events are often outside of the individual's control, and can act as a trigger for episodes of insomnia to occur. Precipitating events can arrive suddenly, such as the death of a spouse. Precipitating events can also build gradually, such as when tensions mount in a failing marriage. Major life events such as retirement or physical illness are important contextual factors to consider because these situations usually affect older adults more so than younger adults (Moos, Brennan, Schutte, & Moos, 2006), and could potentially precipitate an acute episode of insomnia (Morin, Rodrigue, & Ivers, 2003). In addition, there are other significant precipitating factors that are more likely to impact late-life insomnia. For instance, medical conditions (Taylor et al., 2007), medication side-effects (Ancoli-Israel & Ayalon, 2006), and physiological sleep disorders (Smith, Sullivan, Hopkins, & Douglas, 2004) all increase the risk of insomnia. Identifying which specific precipitating factors have occurred will help in further understanding the development of an individual's insomnia problem. In many cases, there are usually a number of interrelated factors that influence the development of insomnia among older adults (Neubauer, 2005; Vitiello et al., 2002). This highlights an added complexity that is involved when understanding sleep problems among older adults.

To further demonstrate the complex nature of sleep problems among older adults, physiologically based sleep disorders, such as obstructive sleep apnoea (OSA), restless legs syndrome (RLS), and periodic limb movement disorder (PLMD) have been found to play a role in influencing insomnia severity (Chung, 2005; Edinger, 2003; Krakow et al., 2001; Lichstein, Riedel, Lester, & Aguillard, 1999; Mucsi et al., 2005; Smith et al., 2004). Physiological sleep disorders are prevalent conditions within the community that increase in

frequency and severity among older adults (Hornyak & Trenkwalder, 2004). OSA occurs when the upper airway is repeatedly obstructed during sleep, which reduces air flow (hypopnea) or stops it (apnoea) (Lavie, Pillar, & Malhotra, 2002; Smith et al., 2004). These repeated episodes lead to interrupted, poor quality sleep, nocturnal oxygen desaturation, and a notable reduction or absence of REM sleep (Ancoli-Israel, 2004; Lavie et al., 2002). The primary symptoms of OSA are loud snoring/gasping for breath and excessive daytime sleepiness, both of which increase with age (Ancoli-Israel, 2004).

Restless legs syndrome, on the other hand, is a sensorimotor disorder described as an urge to move the legs (akathisia), which is usually accompanied by unpleasant leg sensations that impact sleep (Earley, 2003; Hening, 2002; Picchietti & Winkelman, 2005). These sensations are felt deep within the limb(s) and may be described as crawling, restless, or fidgety in nature (Hening, 2002). The symptoms of RLS are invariably worse while resting and are particularly prominent at night prior to sleep onset (Earley, 2003). Individuals with RLS often feel the need to move the legs, which temporarily relieves the discomfort (Earley, 2003).

The majority of older adults with RLS experience periodic limb movements during sleep (PLMS; Hornyak & Trenkwalder, 2004; Montplaisir, Boucher, Poirier, Lavigne, Lapierre, & Lesperance, 1997). PLMS are brief repetitive limb movements which occur whilst asleep (Picchietti & Winkelman, 2005). Periodic limb movement disorder (PLMD) is diagnosed when an individual with PLMS reports insomnia and/or excessive sleepiness, with no other disorder being present to explain the symptoms (Hornyak & Trenkwalder, 2004). The diagnosis of PLMD requires polysomnographic confirmation, while RLS can be diagnosed based on an individual's symptomatology history (Hornyak & Trenkwalder, 2004). Since most individuals with PLMD have RLS, studies have investigated and critiqued the value of PLMD as a diagnostic category (e.g., Nicolas, Lesperance, & Montplaisir, 1998).

Physiological sleep disorders are often comorbid with increased levels of insomnia (Chung, 2005; Krakow et al., 2001; Mucsi et al., 2005; Smith et al., 2004). Studies have reported that the percentage of older adults with OSA and insomnia range from 25% to 50% (Sahai, Staats, & Olsen, 2001; Smith et al., 2004; Lichstein et al., 1999). In addition, Musci et al. (2005) reported that older adults with RLS were twice as likely to have clinically significant levels of insomnia compared to older adults without RLS. Despite these findings, most studies that investigate insomnia have excluded participants with comorbid sleep problems. For example, in Morin et al.'s (1999) influential study that was highlighted earlier, 40 participants were excluded from their original sample of 163 due to having a comorbid physiological sleep disorder. Future research that includes older adults with insomnia and comorbid sleep problems is likely to produce findings that are relevant to a greater proportion of the community. Furthermore, research that investigates individuals with comorbid sleep conditions may add new information to how these conditions are maintained.

Ideally, resolution of the precipitating stressors (e.g., OSA or RLS) should allow an improvement in the insomnia symptoms, however, new perpetuating practices may unintentionally promote continued sleep disturbance independent of the original cause (Glovinsky & Spielman, 2007). Perpetuating practices are maladaptive responses to the initial sleep difficulty that serve to maintain the insomnia problem. For instance, daytime napping to make up for a poor night's sleep can undermine the ability to sleep the next night, leading to a problematic sleep-disruptive cycle (Campbell et al., 2005; Harvey, 2002; Monk et al., 2001). Moreover, the misguided use of night time alcohol or caffeine can increase the likelihood of unintentional sleep disruption during the night (Harvey, 2002). People can also develop intensely-held unhelpful beliefs about sleep, which can make going to bed a negative experience, further interfering with the ability of comfortably initiating and/or maintaining sleep (Espie, 2002; Harvey, 2002, 2004; Morin, 1993; Morin et al., 2002). Ultimately, people

with persistent insomnia endorse maladaptive strategies that transform an acute period of insomnia into a chronic problem.

In summary, several interrelating factors are likely to influence the development and maintenance of insomnia among older adults. To capture these factors succinctly, researchers have grouped potential causes of insomnia into predisposing, precipitating, and perpetuating categories. Characteristics such as a family history of sleep problems may predispose an individual to experience insomnia. Situational stressors or events may precipitate the onset of an insomnia episode, such as the death of a spouse or a physiological sleep disorder (e.g., OSA and/or RLS). Perpetuating factors often include thoughts or behaviours that unintentionally maintain the sleep disturbance, such as napping during the day to compensate for poor sleep the night before.

An increasing amount of research has been dedicated towards investigating the potential consequences of insomnia. Insomnia has been found to have negative consequences in many areas of life (Benca, 2001; Bootzin & Epstein, 2011; Hamilton et al., 2007; Pigeon & Perlis, 2007; Taylor, Lichstein, & Durrence, 2003). For instance, several studies have reported that people with insomnia experience decreased quality of life (e.g., Leger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001; Katz & McHorney, 2002). More specifically, people with insomnia are more likely to report an increased frequency in accidents and falls (Brassington, King, & Bliwise, 2000), and have poorer physical and mental health compared to people without insomnia (Benca, 2001; Taylor et al., 2003). In addition, individuals with insomnia have increased healthcare costs, which translate into a significant societal economic burden (Walsh, 2004). In fact, it has been suggested that insomnia costs the United States of America approximately $30 billion each year (Ozminkowski, Wang, & Walsh, 2007). Currently, the most compelling evidence on the consequences of insomnia has been found in studies investigating the relationship between

insomnia and depression (Manber & Chambers, 2009; Morawetz, 2003; Pigeon & Perlis, 2007).

Insomnia and Depression

There has been a long standing strong association between insomnia and depression (Manber & Chambers, 2009; Mayers & Baldwin, 2006; Morawetz, 2003; Nowell & Buysse, 2001; Pigeon & Perlis, 2007). For instance, studies have reported that up to 90% of individuals with major depression endorse clinically significant levels of insomnia (Perlis, Giles, Buysse, Tu, & Kupfer, 1997; McCall, Reboussin, & Cohen, 2000; Tsuno, Besset, & Ritchie, 2005). Historically, insomnia was viewed primarily as a symptom or a natural consequence of depression (McCall et al., 2000; Nowell & Buysse, 2001; Roth, 2007). Recently, however, researchers (e.g., Buysse et al., 2008; Morawetz, 2003; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005) have begun to reassess the direction of this relationship.

Over the past 20 years, studies have started to explore whether insomnia precedes depression. Although a small collection of studies have questioned the importance of disturbed sleep as a predictor of depression (e.g., Roberts, Shema, Kaplan, & Strawbridge, 2000), convincing evidence has emerged indicating that high levels of insomnia confers an increased risk for the development of depression (e.g., Buysse et al., 2008; Perlis et al., 2006; Pigeon & Perlis, 2007; Riemann & Voderholzer, 2003; Taylor et al., 2005). For instance, as many as 40% of individuals with depression report that insomnia preceded the onset of a first depressive episode, and 56% report that insomnia preceded the recurrence of depression (Ohayon & Roth, 2003). Insomnia has also been associated with increased risk of developing an anxiety disorder or substance disorder (Ohayon & Roth, 2003). Most people, however, with heightened levels of anxiety of substance use reported that insomnia occurred

concurrently with or after the onset of these problems, and not as a prodromal feature as is typical in the development of depression (Ohayon & Roth, 2003).

There are at least 12 known longitudinal studies that have indicated insomnia carries an increased risk of new-onset and recurrent depression over time frames of between 3 months and 3 years (e.g., Ford & Kamerow, 1989; Livingston et al., 1993; Mallon, Broman, & Hetta, 2000; Perlis et al., 2006). In a seminal study conducted by Ford and Kamerow (1989), nearly 8000 participants were examined at baseline and follow-up one year later to investigate whether insomnia increased the risk of depression. The prevalence of insomnia at baseline was 11%. Follow-up assessments found that the risk of developing new cases of depression was nearly 40 times greater when insomnia was present at both interviews, compared to those participants without insomnia. The risk of developing a new episode of depression was much less for those who had insomnia that resolved by follow-up. This was one of the first studies to suggest that insomnia preceded depression, rather than being a consequence of depression.

Reports also show that insomnia can confer depression risk for a period that extends over decades (Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997; Mallon et al., 2000). In Chang et al.'s study, a total of 1053 university students provided information on sleep habits during their course and were re-assessed 30 to 40 years after they graduated. During a median follow-up period of 34 years, 101 participants developed clinical levels of depression and 13 participants ended their own life. It was determined that the participants who reported high levels of insomnia during their period at university were twice as likely to develop depression compared to participants with low levels of insomnia (Chang et al., 1997). These findings suggest that high levels of insomnia are indicative of a greater long-term risk for subsequent clinical depression.

The relationship between insomnia and depression appears to also depend on the severity of the insomnia (Hohagen et al., 1993; Katz & McHorney, 1998; Lustberg & Reynolds, 2000). Hohagen et al. were first to investigate whether the level of insomnia influenced depression severity in more than 2500 participants from a primary care setting. Assessments were collected at baseline, four months, and two years. It was reported that the presence of depression at the two year follow-up was much stronger if participants presented with severe levels of insomnia at baseline. Similar results were also found in an American study involving 1800 participants over a two year period (Katz & McHorney, 1998). At baseline, 16% presented with severe levels of insomnia, and 34% with mild insomnia. At the two year follow-up assessment, 59% of those with mild insomnia, and 83% of those with severe insomnia, still had sleep problems. Katz and McHorney found that participants with mild levels of insomnia were 2.6 times at risk of developing depression, and participants with severe levels of insomnia were 8.2 times at risk of developing depression, compared to those who did not present with insomnia problems. It was concluded that individuals with severe levels of insomnia were at particular risk of developing future depression.

Studies that have investigated the relationship between insomnia and depression mainly focus on adults under the age of 65 years. To increase the evidence base for older adults, Perlis et al. (2006) collected data from an American community sample of healthy older adutls (age range 60-94 years) to assess the extent to which insomnia predicted depression. Participants were evaluated at two time points separated by a one-year interval. Out of the 147 participants, 45% were classified as not having insomnia, 32% had indeterminate (sub-clinical) insomnia, and 23% had persistent (clinical) insomnia at the beginning of the study. Twelve participants developed depression during the one-year follow-up period. Of these twelve, two did not have insomnia, four had indeterminate insomnia, and six had persistent insomnia. As a result, participants with persistent insomnia were

approximately six times more likely to develop a first episode of depression, compared to participants without insomnia. It was evident that older adults with persistent insomnia were at an increased risk of depression compared to younger adults, since the relative risk estimate in Perlis et al.'s study was higher compared to studies that explored younger adults.

Despite Perlis et al.'s (2006) convincing results, there were some important limitations worth noting. In Perlis et al.'s study, insomnia was assessed using the Structured Clinical Interview for the *DSM-IV-TR* (SCID; APA, 2000). Although this is a well known clinical diagnostic assessment tool, it is not a sleep-specific measure that explores an individual's level of insomnia. The Insomnia Severity Index (ISI; Bastien et al., 2001), for example, may have provided a better assessment of insomnia severity. Another key limitation inherent within Perlis et al.'s study was its small sample size. Unlike earlier studies (e.g., Chang et al., 1997; Livingston et al., 2000; Mallon et al., 2000), Perlis et al.'s estimate of risk appeared to be significantly greater (approximately 6.0 vs. 3.0) compared to other studies. Although this result may represent a genuine increased risk among older participants, the estimated risk may also have been simply a statistical artefact owing to the small number of cases in each insomnia group. Perlis et al. suggested that future studies need to obtain larger sample sizes, which would likely increase the participants in each comparison group and make for more compelling results.

In summary, an increasing body of research has been dedicated to investigating the relationship between insomnia and depression. There is a strong relationship between insomnia and depression, with higher levels of insomnia being associated with higher levels of depression. Historically, it was believed that insomnia was primarily a symptom or natural consequence of depression. Recent data, however, clearly demonstrate that insomnia often precedes the onset of depression. Convincing longitudinal evidence has shown that participants with high levels of persistent insomnia are approximately three to six times more

likely to develop depression compared to participants without complaints of insomnia, particularly among older adults. Although it has been determined that increased levels of insomnia predict depression severity, the mechanisms underlying this relationship are unclear and require further exploration.

There has been little research attempting to explain how insomnia influences depression. A small collection of studies have begun to explore this relationship from a neurobiological perspective. The neurobiological point of view suggests neuroendocrine imbalances associated with persistent insomnia may directly or indirectly predispose an individual to develop depression (Adrien, 2002; Monteleone & Maj, 2008; Nofzinger et al., 2005). These neuroendocrine abnormalities may, in turn, represent biological factors that make insomnia a risk factor for depression. For instance, one study investigated how neurobiological abnormalities associated with insomnia influenced depression severity (Nofzinger et al., 2005). The study included 29 participants with major depression and 28 healthy participants with no mental disorder. Participants completed electroencephalographic sleep and regional cerebral glucose metabolism assessments during both waking and NREM sleep. It was found that participants with depression showed smaller decreases than healthy participants in relative metabolism in broad regions of the frontal, parietal, and temporal cortex from waking to NREM sleep. In addition, participants with depression showed larger decreases than healthy participants in relative metabolism in the left amygdala, anterior cingulate cortex, cerebellum, parahippocampal cortex, fusiform gyrus, and occipital cortex. Overall, Nofzinger and colleagues suggested that insomnia was associated with depression through a decrease in metabolic activity during NREM sleep.

Another study from the neurobiological perspective proposed that the serotoninergic system was responsible for the relationship between insomnia and depression (Adrien, 2002). Adrien suggested that acute periods of sleep deprivation have antidepressant effects to certain

neurotransmitters (e.g., serotonin). Thus, short-term sleep loss may represent an attempt to counteract serotoninergic hypofunctioning in individuals at risk of developing depression, and in that sense be part of a neurobiological self-therapeutic mechanism. When acute sleep loss turns into chronic insomnia, however, Adrien proposed that the overall serotonergic deficit becomes profound via changes in REM sleep and a decrease in serotonergic tone, resulting in possible increased severity of depressive symptoms. While such a proposal is entirely speculative at present, Adrien suggested that change in the serotoninergic system was one possible mechanism by which insomnia could influence depression. Although studies such as Nofzinger et al. (2005) and Adrien (2002) have begun to offer a neurobiological explanation of how insomnia influences depression, some important issues are worth highlighting.

Three central limitations emerge from the neurobiological model of insomnia and depression. The first involves an inconsistency in the neurobiological pathways in which insomnia influences depression. For instance, Nofzinger et al. (2005) reported that insomnia was related to depression through a decrease in metabolic activity during NREM sleep. On the other hand, Adrien (2002) suggested that the serotoninergic system was responsible for the relationship between insomnia and depression via changes in REM sleep. These major differences in results indicate that there is a discrepancy in how models of insomnia influence depression from the neurobiological perspective.

A second key limitation involves the relationship between the neurobiological conceptualisation of insomnia and depression and the implications this model has for future treatment. Since the neurobiological perspective has a medical underpinning, researchers from this perspective suggest that insomnia and depression should be treated with medication (e.g., Adrien, 2002; Benca, 2005; Monteleone & Maj, 2008). This is a contentious recommendation because sleep medication can often have addictive properties (Holbrook,

Crowther, Lotter, Cheng, & King, 2000; Mendelson et al., 2004), meaning that an individual may become dependent on the medication and require an increasing dose to experience the same therapeutic effect. Also, psychotropic medication can have severe side effects, and has the potential to exacerbate sleep/mood problems (Mendelson et al., 2004; Ohayon et al., 1999). Furthermore, symptoms of insomnia and depression can re-appear once an individual stops taking medication, resulting in poor long-term treatment effects from this form of treatment (Dikeos & Soldatos, 2002). Therefore, the applicability of a neurobiological explanation of how insomnia relates to depression is problematic due to its medically-based treatment implications.

The final limitation involves a failure of the neurobiological model to account for the cognitive elements that may serve to explain the relationship between insomnia and depression. This is a major oversight because the conceptualisation of insomnia currently has a strong psychological empirical foundation (e.g., Edinger et al., 2001; Espie et al., 2000; Harvey, 2002, 2005; Morin, 1993), and this cognitive understanding of insomnia translates directly into practical treatment options (Buysse, 2004; Morawetz, 2003; Morin, 2004). In fact, it has been recommended that one of the goals in the psychological treatment of insomnia is to help individuals cease medication use (Morawetz, 2003), which runs against the implications made from the neurobiological perspective. Overall, the three central limitations outlined above demonstrate that the neurobiological perspective alone is not sufficient to explain how insomnia impacts depression.

Currently there are no studies that have explored how insomnia influences depression from a cognitive perspective. Identification of psychological variables that contribute towards insomnia and depression is important because the results are likely to further the understanding, prevention, and treatment of these problems. One avenue that insomnia may influence depression is through an individual's dysfunctional beliefs about sleep.

Dysfunctional Beliefs About Sleep

Dysfunctional beliefs about sleep refer to unhelpful sleep-related cognitions that can potentially heighten insomnia severity (e.g., 'When I have trouble getting to sleep, I should stay in bed and try harder'; Morin, 1993). Although the name ‘dysfunctional beliefs about sleep’ implies that the presence of such beliefs is inherently 'dysfunctional', it is more accurate to convey that strong or rigid endorsement of these beliefs can be unhelpful or maladaptive (Carney & Edinger, 2006). As such, individuals with low levels of insomnia would not be expected to completely disagree with these sleep-related beliefs, instead, their degree of agreement would be moderately low and reflect some flexibility in the beliefs (Carney & Edinger, 2006). In contrast, strong endorsement of these beliefs about sleep may represent less flexibility and, consequently, more distress when faced with situations that appear to confirm such beliefs (Carney & Edinger, 2006).

Cognitive conceptualisations of insomnia posit that rigidly held or self-defeating beliefs and attitudes about sleep play an important role in maintaining insomnia (Carney & Edinger, 2006; Harvey, 2002, 2005; Morin, 1993; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). It has been reported that approximately 80% of individuals suffering insomnia experience rigidly held unhelpful beliefs about sleep (Morin, 1993; Morin et al., 1993). Evidence has shown that high levels of insomnia are correlated with more strongly endorsed maladaptive beliefs about sleep (Edinger et al., 2001; Espie et al., 2000; Morin, 1993; Morin et al., 1993; Morin, Vallieres, & Ivers, 2007). In addition, research has illustrated that some unhelpful sleep-related beliefs are held more rigidly among older adults (Ellis, Hampson, & Cropley, 2007). For example, the belief that changes in sleep patterns with ageing are pathological can cause particular psychological distress among older adults (Ellis et al., 2007). Likewise, the expectation that eight hours of sleep is essential to function adequately during the day can produce performance anxiety, especially when this sleep

duration is not met (Ellis et al., 2007). Such faulty appraisal may either turn what were normal changes in sleep patterns among older adults into a clinical problem, or prolong what might have otherwise been a situational sleep disturbance into a chronic problem (Ellis et al., 2007; Morin, 1993, 1994).

Morin et al. (1993) conducted a pioneering study that explored whether older adults with insomnia experienced different sleep-related cognitions compared to older adults without insomnia. This study examined the beliefs and attitudes about sleep among 145 older adults. Morin et al. used a structured clinical interview to divide participants into two separate groups, participants with insomnia (n = 74) and participants without insomnia (n = 71). Participants completed the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS; Morin, 1993, 1994), consisting of 30 statements concerning various beliefs, expectations, and attributions about several sleep-related themes (Morin et al., 1993). The results indicated that participants with insomnia endorsed more strongly dysfunctional beliefs about sleep, relative to participants without insomnia. More specifically, participants with insomnia endorsed stronger beliefs about the negative consequences of insomnia, expressed more hopelessness about the fear of losing control of their sleep, and more helplessness about its unpredictability compared to participants without insomnia. These findings suggested that rigidly held dysfunctional beliefs about sleep could be maladaptive in nature and be instrumental in influencing and perpetuating insomnia.

It is commonly accepted that individuals with high levels of insomnia often display unhelpful sleep-related cognitions (Carney & Edinger, 2006; Espie, 2002; Harvey 2002; Morin, 1993). Few studies, however, have investigated the role of dysfunctional beliefs about sleep among people with insomnia and comorbid physiological sleep conditions. A possible reason for this lack of research could be due to physiological sleep problems being primarily understood and treated from a medical perspective.

There are only two known studies that have investigated the role of beliefs about sleep in the physiological sleep research (Edinger, 2003; Smith et al., 2004). Smith et al. explored the prevalence of insomnia in a consecutive series of 105 Australian older participants (mean age of 53 years) presenting to a sleep unit for investigation of suspected OSA. The authors assessed factors that may impact OSA and insomnia, including sleep-related beliefs. Participants underwent a polysomnogram diagnostic study and completed a battery of sleep measures (e.g., Dysfunctional Beliefs and Attitudes About Sleep 10-Item Scale, DBAS-10). Two groups were formed, which comprised of those with OSA only (n = 64) and those with comorbid OSA and insomnia (n = 41). Results showed a strong positive correlation between OSA and insomnia symptom severity. Findings related to beliefs about sleep indicated that both groups had a high level of dysfunctional sleep cognitions. More specifically, participants in the OSA-insomnia group reported more rigid dysfunctional beliefs about sleep and greater disruption in habitual sleep than those with OSA only. Furthermore, the relationship between insomnia and unhelpful sleep-related beliefs was stronger among people with OSA and insomnia compared to people only OSA. Smith et al. concluded that there was a strong positive correlation between OSA and insomnia, and individuals with both conditions tended to experience stronger dysfunctional beliefs about sleep than individuals with OSA only.

A second study also found that individuals with physiological sleep disorders endorse high levels of dysfunctional beliefs about sleep (Edinger, 2003). Edinger explored whether participants with insomnia and comorbid sleep disorders (RLS and periodic limb movements, PLM) displayed differing levels of dysfunctional beliefs about sleep compared to individuals without sleep problems. Archival data was obtained from a sleep history questionnaire, polysomnogram, and sleep diary. Based on these measures, participants were placed into one of three groups (RLS/PLM, insomnia, or no sleep problems). Statistical comparisons showed that the RLS/PLM and insomnia groups differed significantly from the control group in

relation to their level of dysfunctional beliefs about sleep. This result suggested that cognitive factors, such as sleep-related beliefs, may play a role in comorbid sleep conditions. The findings from Smith et al. (2004) and Edinger's (2003) studies were the first to include cognitive elements within the physiological sleep disorder research. Since the cognitive mechanisms of sleep have largely been ignored in the physiological sleep field, further studies are needed to explore the role of dysfunctional beliefs about sleep in individuals with insomnia and physiological sleep disturbances.

In summary, sleep-related maladaptive cognitions play an important role in perpetuating and exacerbating insomnia. This is particularly relevant for older adults because certain dysfunctional beliefs about sleep are endorsed more strongly among this age group (Ellis et al., 2007). Exploratory studies have reported that individuals with physiological sleep disorders (i.e., OSA, RLS) exhibit stronger levels of dysfunctional beliefs about sleep compared to individuals without these conditions (Edinger, 2003; Smith et al., 2004). These findings collectively indicate that individuals with chronic sleep problems often endorse inflexible dysfunctional beliefs about sleep.

A recently developed cognitive model (Harvey, 2002, 2005) has highlighted how cognitive mechanisms, such as dysfunctional beliefs about sleep, perpetuate insomnia. Harvey proposed that insomnia was maintained by several interrelating cognitive processes that operate at night and during the day. These cognitive processes include negatively toned cognitive activity, arousal/distress, selective attention, misperception, dysfunctional beliefs about sleep, and safety behaviours. Recognising how these cognitive elements influence the maintenance of insomnia may assist in understanding the relationship between insomnia and depression.

Cognitive Model of Insomnia

Harvey's cognitive model of insomnia (2002, 2005) was based on the work pioneered by cognitive theorists such as Aaron Beck (e.g., Beck, 1976), as well as from evidence that identified the importance of cognitive processes to insomnia (e.g., Morin, 1993). The first aspect to Harvey's cognitive model proposed that people with insomnia lie in bed worrying about a variety of topics, including not being able to fall asleep. Harvey suggested that people with insomnia go to bed preoccupied with needing to get to sleep quickly and wanting to obtain as much sleep as possible. These thoughts are likely to be accompanied by concerns about health, concerns about not coping, and thoughts relating to unresolved past, present, and future issues (Harvey, 2000; Wicklow & Espie, 2000). As a result, Harvey proposed that people who develop problems with insomnia experience excessive negatively toned cognitive activity during the pre-sleep period.

The cognitive model specifies that this initial stage of excessive cognitive activity activates the sympathetic nervous system, thereby triggering autonomic physiological arousal and emotional distress (Harvey, 2002; Espie, 2002; Jansson & Linton, 2007). While activation of the sympathetic nervous system was an important response to threats faced by mankind in the past (e.g., to escape danger for survival), Harvey reported that activation of this system is unhelpful when the state of threat is generated by excessively toned negative worry while trying to sleep. This combination of excessive cognitive activity, autonomic arousal, and emotional distress propels the individual into a state that negates sleep onset or sleep maintenance.

Harvey (2002) suggested that when an individual's level of arousal and distress become heightened, there is a narrowing of attention toward sleep-related threats that can be related to internal stimuli (e.g., bodily sensations) and/or external stimuli (e.g., noise). It is proposed that people with insomnia selectively attend to and monitor for sleep-related cues,

such as bodily sensations that inhibit the ability to fall asleep, or checking the bedside clock constantly to calculate how long it is taking to fall asleep (Harvey, 2000; Harvey & Schmidt, 2000). This selective attention and monitoring is usually automatic in the sense that it consumes minimal attention resources and can happen without conscious decision making. As selective attention increases the chance of detecting random cues that can be misinterpreted, constant cognitive monitoring is likely to provide further cause for worry.

The following phase in Harvey's (2002) cognitive model of insomnia includes misperception. This involves people with insomnia exhibiting a distorted perception of the amount of sleep they actually obtain (Bonnet & Arand, 1994; Harvey, 2005; Tang & Harvey, 2004). For instance, an individual with insomnia may overestimate how long it takes to fall asleep and/or underestimate how long they sleep in total. Harvey suggests that insomnia is like most other psychological disorders in being characterised by 'distortions in reality' (Beck, 1976, p. 218). To give some examples of misinterpretation in other psychological disorders, Harvey (2005) suggested that people with anorexia nervosa think they are overweight when actually, they are underweight; and people with panic disorder think they are having a heart attack when actually they are experiencing symptoms of anxiety. In a similar way, it is possible that unhelpful cognitive processes are operating that mislead individuals with insomnia into overestimating the extent to which their sleep is inadequate (Harvey, 2002, 2005).

One of the most important aspects to Harvey's (2002) cognitive model of insomnia involves the endorsement of dysfunctional beliefs about sleep. As was previously discussed in Morin's research, strongly held maladaptive sleep beliefs are a focal part of the insomnia maintenance cycle (Morin, 1993; Morin et al., 1993; Morin, 2004). Harvey proposed that dysfunctional beliefs about sleep are closely interrelated with the preceding cognitive processes in the model (negatively toned cognitive activity, arousal/distress, selective

attention, misperception), and serve to exacerbate these unhelpful cognitive processes. For example, an individual who is experiencing persistent negatively toned cognitive activity at night is likely to develop a strongly held belief that they need to stay in bed and try harder to fall asleep (Harvey, 2002). In other words, the unhelpful cognitive mechanisms that occur during the pre-sleep period increase the intensity of the maladaptive sleep-related beliefs. Consequently, the endorsement of such a belief contributes further to psychological and physiological distress during the pre-sleep period, consolidating the interrelated maladaptive cognitive cycle that induces insomnia.

Harvey (2002) proposed that the more iteration surrounding the dysfunctional cognitive cycle, the more likely a real sleep deficit will occur. For instance, escalating cognitive activity, physiological arousal/distress, and intense monitoring of sleep-related cues are not conditions that are conducive to sleep onset (Ansfield, Wegner, & Bowser, 1996; Harvey, 2002; Jansson & Linton, 2007). A study reported by Ansfield et al. provided support for the proposal that increasing cognitive load compromises sleep. Ansfield et al. instructed good sleepers without insomnia to either fall asleep as quickly as they could or to fall asleep whenever they desired under high (loud marching music) or low cognitive load (soft relaxation music) conditions. The group given the most cognitively demanding task (high load plus instructions to fall asleep quickly) had the most difficulty falling asleep. This finding demonstrates how people who are caught in excessive cycles of cognitive activity during the pre-sleep period are likely to develop a real sleep deficit.

In an attempt to cope with the escalating worry influenced by the dysfunctional cognitive processes, people with insomnia often make use of safety behaviours (Harvey, 2002, 2005; Salkovskis, 1989, 1996). Harvey (2002) described safety behaviours as strategies that people with insomnia develop in an attempt to avoid or prevent a feared outcome, typically a fear of not being able to fall asleep. Unfortunately, many such strategies impede

natural self-corrective processes and further strengthen dysfunctional beliefs about sleep (Ree & Harvey, 2004; Roehrs & Roth, 2001). For example, drinking alcohol at night may act as a safety behaviour because it may promote sleep onset, however, it results in more awakenings and more disturbed sleep during the night (Roehrs & Roth, 2001). In addition, people with insomnia may try to suppress or control their thoughts during the pre-sleep period to reduce their level of cognitive activity. Studies have suggested that these safety behaviours have a paradoxical effect, which increases unwanted cognitive activity and lengthens sleep onset latency (Gendron, Blais, & Morin, 1998; Harvey, 2001). Thus, safety behaviours unintentionally maintain unhelpful beliefs about sleep and make the feared outcome more likely to occur.

In summary, Harvey's (2002, 2005) cognitive model of insomnia highlights the close relationship between insomnia and dysfunctional cognitive processes. It is proposed that individuals who suffer from high levels of insomnia tend to be preoccupied about their sleep and about the daytime consequences of not getting enough sleep. This excessive negatively toned cognitive activity activates both autonomic arousal and emotional distress. Cognitive arousal triggers selective attention and monitoring of internal and external sleep-related threat cues. These processes mislead the individual into overestimating the extent of the perceived sleep deficit. Together, individuals develop rigidly held dysfunctional beliefs about sleep that are a central component within the insomnia maintenance cycle. Finally, counterproductive safety behaviours may further exacerbate the dysfunctional cognitive processes resulting in persistent problems with insomnia. Hidden within Harvey's cognitive model lies an important psychological construct that could further enhance the understanding of how unhelpful sleep-related cognitions relate to depression.

Emerging from Harvey's cognitive model of insomnia (2002) is the concept of hopelessness. The term hopelessness refers to having a negative attitude towards the future

(Beck & Steer, 1988). Individuals that possess a high sense of hopelessness believe they cannot control circumstances in their lives and tend to experience a pervasive negative bias about the future (Beck & Steer, 1988; Seligman, 1975). This concept of hopelessness is central to those individuals who experience rigidly held dysfunctional beliefs about sleep. That is, the more strongly an individual endorses inflexible sleep cognitions, the greater their perception of loss of control over their ability to sleep.

The relationship between insomnia, unhelpful sleep beliefs, and hopelessness was briefly commented on in Morin et al.'s (1993) original study, where participants with insomnia were found to endorse stronger beliefs about the negative consequences of insomnia, express more hopelessness about the fear of losing control of their sleep, and experience more helplessness about its unpredictability compared to participants without insomnia. Surprisingly, few studies have expanded upon Morin et al.'s finding to investigate further how dysfunctional beliefs about sleep relate to hopelessness. While Harvey's (2002) cognitive conceptualisation of insomnia did not explicitly discuss this relationship, it becomes evident from the model how an individual with insomnia could develop a sense of hopelessness via strongly held dysfunctional beliefs about sleep. Reviewing the unhelpful cognitive processes that operate during the day in Harvey's model will help identify how sleep-related cognitions could influence hopelessness.

A novel feature of Harvey's (2002) insomnia model is that daytime cognitive processes are assumed to be of equal significance to the maladaptive processes that operate at night. Harvey proposed that parallel mechanisms to those described at night also operate during the day. This exchange of night and day dysfunctional cognitive processes is where an underlying sense of hopelessness could develop. For instance, immediately upon waking, a person with insomnia is likely to appraise the quality and amount of sleep they obtained (Bonnet & Arand, 1994; Harvey, 2004; Tang & Harvey, 2004). This is a particularly

vulnerable period for an individual with insomnia due to the hypnopompic state (otherwise referred to as sleep inertia), which involves a 'vagueness' in cognitive functioning immediately upon waking (Dinges, 1990). Given that people with insomnia tend to automatically underestimate the amount of sleep they obtain and usually engage in the appraisal process of sleep when they wake in the morning (Bonnet & Arand, 1994; Clark, 1999; Harvey, 2004; Tang & Harvey, 2004), it is likely that the person will go into the day with the belief that they did not get enough sleep. These automatic thoughts will be particularly acute if the perceived sleep debt has been accumulating over several nights (Harvey, 2004). Consequently, it is likely that a person with persistent insomnia will be preoccupied with sleep-related topics such as feeling fatigued and tired, not coping, and not performing adequately during the day. Additional daytime thoughts may include concerns about losing control of their ability to sleep and falling ill as a result of inadequate sleep, which in turn could increase a sense of negativity toward the future. This excessively toned negative activity provides an example of how persistent sleep problems could influence an individual to develop a sense of hopelessness.

Several more examples can be drawn from Harvey's (2002, 2005) cognitive model of insomnia that demonstrates how hopelessness could result from strongly held sleep-related beliefs. Harvey suggested that individuals with insomnia who selectively attend and intensely monitor their environment are likely to increase the chance of detecting ambiguous cues (e.g., feelings of tiredness in the legs) that can then be misinterpreted (e.g., "I mustn't have slept enough last night"), leading to misperception of other daytime events (e.g., "I won't be able to cope today"). The unhelpful interpretation of seemingly minor events could occur numerous times during the day and night. These negative experiences are likely to build up over time because the dysfunctional beliefs remain unchanged or disproven, which possibly contributes towards an increased sense of hopelessness. This cascade of dysfunctional cognitive

processes could consolidate a pessimistic view about the future, as their sleep continues to be disrupted.

Harvey (2002) also reported that the use of safety behaviours can contribute to distress and worsening sleep, as well as preventing disconfirmation of dysfunctional beliefs about sleep. For example, cancelling a social engagement could maintain the belief that "I'm too tired to go out", yet the endorsement of this belief may fuel a sense that life is hopeless due to persistent poor sleep. Another example includes an elderly individual who believed they needed at least 8 hours of continuous sleep in order to cope the following day to remain healthy. Despite the individual's best efforts, 8 hours of continuous sleep was rarely managed. It is possible that the individual's daytime functioning and health became a major source of worry because the endorsement of this belief remained consistently strong. As a result, the persistent inflexible nature of the belief could increase the older adult's level of hopelessness, since the individual continued to not meet the goal of being able to sleep 8 continuous hours per night. These examples illustrate how a sense of hopelessness could develop among people with high levels of dysfunctional beliefs about sleep. Although the relationship between dysfunctional beliefs about sleep and hopelessness can make theoretical sense with reference to Harvey's cognitive model, research needs to be conducted to empirically validate this assertion.

The relationship between dysfunctional beliefs about sleep and hopelessness could be a vital area of future research because these cognitive mechanisms could provide a psychological pathway that explains how insomnia influences depression. Since Harvey's cognitive model of insomnia was heavily influenced by Beck's theory of depression (e.g., Beck, 1963, 1967, 1976; Beck, Rush, Shaw, & Emery, 1979), reflecting upon Beck's research will assist to further the understanding how psychological mechanisms influence the relationship between insomnia and depression.

Cognitive Theory of Depression

Cognitive models, such as Harvey's (2002) model of insomnia and Beck's (1976) model of depression, share the premise that maladaptive thinking and negative appraisals of events contribute to the development and perpetuation of insomnia and depression. Beck's cognitive model postulates that three psychological concepts explain the development of depression, including the cognitive triad, schemas, and cognitive errors. Each of these concepts can provide further theoretical support for how dysfunctional beliefs about sleep relates to hopelessness.

Beck's (1963, 1976) cognitive triad consists of three cognitive patterns that bias an individual to think in a negative manner. The first element of the triad revolves around an individual's negative view of the self (self-criticalness). Beck reported that the individual tends to attribute unpleasant experiences to a psychological, moral, or physical deficit of the self. This influences the individual to see the self as inadequate, undesirable, and worthless. The individual tends to underestimate or criticise the self and believes they lack the attributes needed to attain happiness, contentment, or resolution.

The second element of Beck's (1976) triad involves the tendency to interpret ongoing experiences in a negative way (general negativity). The individual views the world as making exorbitant demands on the self and/or presents insurmountable obstacles to reaching life's goals. Constant misinterpretations between the self and environment occur, causing the individual to perceive the self as defective or inadequate. Consequently, the individual's initial interpretation is negatively biased to tailor pieces of information to fit their negative conclusion.

The final element of Beck's (1976) cognitive triad is particularly relevant to the association between maladaptive sleep beliefs and hopelessness. This element involves an individual anticipating that their current difficulties will continue indefinitely (hopelessness).

The individual makes long-term projections based on their current circumstances, expecting that there will be unremitting hardship, frustration, and despair. When the individual considers undertaking a specific task in the immediate future, they expect to fail. For example, an individual that consistently experiences sleep difficulties may strongly endorse a belief that their sleep will never improve, and in turn, a sense of hopelessness could develop. Thus, Beck's cognitive triad demonstrates how dysfunctional beliefs about sleep could be related to hopelessness.

The second component in Beck's (1976) model consists of the concept of schemas. When a person is confronted with a particular situation, a schema related to the circumstance is activated. Beck stated that schemas are the basis for moulding neutral pieces of information into thoughts or cognitions that develop into emotions. Schemas therefore constitute the basis for screening out, differentiating, and coding stimuli that confront individuals (Beck et al., 1979). Although it is common that people may interpret the same situation in different ways, Beck proposed that an individual tends to develop consistencies in their responses to similar types of events. For example, an individual who continues to find it difficult to initiate sleep could develop unhelpful schemas that are activated during the pre-sleep period. As a result, relatively stable cognitive patterns form the basis for the regularity of interpretations of a particular set of situations (e.g., attempting to fall asleep), which could develop increased levels of hopelessness due to unhelpful schemas becoming activated in sleep-related situations.

Beck et al. (1979) proposed that the kinds of schemas employed determine how an individual will structure different experiences. The schemas activated in a specific situation directly determine how the person responds. In people with insomnia, for example, individuals' interpretations of sleep-related situations are distorted to fit the prominent dysfunctional schemas. The matching of an appropriate schema to a particular sleep-related

stimulus is disrupted by the intrusion of these overly active idiosyncratic schemas. As these schemas become more active, they are evoked by a wider range of stimuli which are less logically related to them. That is, schemas activated by sleep-related stimuli could expand into more general areas of life. Consequently, the individual loses much of their voluntary control over their thinking processes and is unable to invoke other more balanced schemas to a wider range of situations.

Cognitive errors were the third important component in Beck's (1976, 1979) theory of depression. Beck found that individuals with depression distort reality in a systematic manner that results in a bias against the self and their environment. Beck outlined several cognitive errors that demonstrate faulty information processing. For example, absolutistic thinking (otherwise known as black/white thinking), refers to a tendency to place all experiences in one of two opposite categories (e.g., good verses bad). Cognitive errors are inherent within dysfunctional beliefs about sleep. For instance, an individual that experiences consecutive poor nights' sleep may develop a strongly held belief that "I'm a bad sleeper", demonstrating an example of an absolutistic cognitive error. The result of these cognitive processing errors is that they lead to overly negative and pessimistic interpretations, evaluations, and appraisals of the self and their current context.

In relation to cognitive errors, Beck et al. (1979) proposed that people who are at risk of developing depression are prone to structure their experiences in relatively 'primitive' ways. They tend to make broad global judgements regarding events that impinge on their lives. The meanings that flood their consciousness are likely to be extreme, categorical, absolute, and judgemental. According to this schematic representation, Beck observed that people at risk of depression tended to view their experience as total defeats (non-dimensional) with irreversible consequences (fixed). Concomitantly, these individuals categorise themself as a 'loser' (judgemental) and 'doomed' (irreversible character deficits).

The emotions connected with these thoughts, thus, tended to be negative and possess a heightened sense of hopelessness and depression.

In summary, Harvey's (2002) cognitive model of insomnia was heavily based on Beck's (1963, 1976) cognitive theory of depression. Both models identified that negative cognitive patterns were a central part of these problems. The concept of hopelessness was inherent within Beck's cognitive model, which specifically outlined that individuals at risk of depression experienced an intolerable view towards the future. The concept of hopelessness, however, has rarely been discussed in the insomnia literature. With reference to Harvey's model, chronic insomnia could increase an individual's sense of hopelessness via rigidly held dysfunctional beliefs about sleep. For instance, people who experience debilitating sleeplessness tend to consistently endorse inflexible maladaptive sleep beliefs. Over time, these individuals learn that whatever strategies they are implementing to improve their sleep is not working, thereby creating an increased sense of hopelessness. Beck and Harvey's cognitive models provide a theoretical understanding of how dysfunctional beliefs influence hopelessness. More research is clearly warranted to assess whether dysfunctional beliefs about sleep are related to increased levels of hopelessness.

Hopelessness Theory of Depression

Exploring further the role of hopelessness will strengthen the understanding of how cognition and depression are related. Hopelessness is a psychological construct that gained momentum in the late 1960s and early 1970s, when Seligman and colleagues (1967, 1975) conducted experiments with dogs. Overmier and Seligman (1967) conducted a study in which dogs were subjected to repeated electrical shocks that were not contingent upon their behaviour. During inescapable exposure to electrical shocks, dogs learned that shocks were independent of any responses. These dogs showed striking deficits when placed later in a box

in which a simple act of crossing a barrier would have terminated a new shock. Unlike dogs not previously exposed to uncontrollable shocks, these dogs seemed to develop a sense of helplessness. For example, these dogs showed minimal attempts to escape the shock and were not likely to follow an occasionally successful response. It was determined that helplessness in these dogs was marked by cognitive, emotional, and motivational deficits. Overmier and Seligman called this phenomenon 'learned helplessness', and proposed that helplessness could explain how increased levels of depression arise in humans.

Some key limitations were identified in Overmier and Seligman's (1967) study that paved the way for a reformulated theory of helplessness (Abramson, Seligman, & Teasdale, 1978). This updated theory was known as the explanatory or attribution style theory of helplessness, and proposed that an uncontrollable event did not always cause helplessness (Abramson et al., 1978). It was suggested that individual differences were responsible for this finding, thus, the theory was revised to include a cognitive component. Specifically, an individual's interpretation of an event was seen as a critical determinant in the development of helplessness and depression, rather than simply the objective uncontrollable nature of an event (Abramson et al., 1978). This model proposed that individuals tending to helplessness and depression interpret events as internal (e.g., "It's all my fault"), stable ("Things can't change"), and global (e.g., "It is going to affect everything I do"). According to this theory, identification of individuals at risk of depression should be possible by the way they internally interpret events. As a result, it was suggested that individual differences in explanatory style to events emerged as an important component in the development of depression.

Soon after the revised theory of helplessness was presented, critics argued that the helplessness models contained additional limitations. It was identified that these theories lacked predictive power because they did not specify the conditions under which a certain

causal attribution would be more probable than another (Henkel, Bussfeld, Moller, & Hegerl, 2002). This meant that researchers were uncertain as to whether helplessness/hopelessness preceded the course of depression, rather than just being a natural part of depression. In addition, the role of individual internal verses external responses to events was unclear (Costello, 1972; Henkel et al., 2002). For instance, it was suggested that people with depression often related the causes of their depression to outside forces (Costello, 1972), whereas the revised helplessness theory proposed that people internalised their experiences (Abramson et al., 1978). To address these concerns, Abramson, Metalsky, and Alloy (1989) built on this body of literature to construct the hopelessness theory of depression that is still applied in current research.

The hopelessness theory of depression (Abramson et al., 1989) has addressed a number of limitations that were evident in the preceding helplessness theories. Abramson et al. postulated that the negative attribution style is an important risk factor for a specific constellation of depressive symptoms, which was termed 'hopelessness depression'. In this theory, hopelessness was viewed as a subset of helplessness. This meant that if hopelessness occurs then helplessness also occurs, but not vice versa. It was also hypothesised that there would be an association between negative attribution style and depression symptoms only in the presence, but not in the absence of negatively perceived life events.

The hopelessness theory of depression proposed that individuals with a pessimistic explanatory style tend to attribute the causes of negative events to global and stable factors, whereas individuals with an optimistic explanatory style exhibit the tendency to attribute such causes to specific and unstable factors (Abramson et al., 1989). It was suggested that individuals with a pessimistic explanatory style are also more likely to make depressive inferences about the causes of negative events than individuals without this style. Such consistent inferences increase the likelihood of hopelessness, and once hopelessness

develops, depression is inevitable, since hopelessness is viewed as a proximal and sufficient cause of depression in this theory (Abramson et al., 1989).

The hopelessness theory of depression elaborates on the reformulated learned helplessness theories by delineating two additional cognitive styles that play a role in the etiology of depression. Individuals with pessimistic cognitive styles about consequences and the self tend to view negative events as having many disastrous consequences and view the self as flawed and deficient following negative events (Abramson et al., 1989). When faced with a negative event, individuals with pessimistic explanatory style make attributions that lead them to see the future as hopeless and make it likely they will develop depression. In contrast, individuals with optimistic cognitive styles tend to infer that negative consequences will not follow from negative events and believe that negative events in their life do not mean that they are flawed in any way.

Numerous studies have supported the hopelessness theory of depression (e.g., Alloy et al., 2006; Golin, Sweeney, & Shaeffer, 1981; Isaacowitz and Seligman, 2001; Metalsky, Joiner, Hardin, & Abramson, 1993). An early study of college students found that global and stable causal inferences for negative events were predictive of depressive symptoms 1 month later even after controlling for initial depression scores (Golin et al., 1981). Isaacowitz and Seligman (2001) recruited community-dwelling older adults to investigate whether hopelessness continued to be a risk factor later in life. Consistent with other findings, hopelessness predicted depressive symptoms 1 year later. One study found that those with negative cognitive styles were 3.5 time more likely to develop a minor depressive episode, and were 6.7 times more likely to develop a major depressive episode (Alloy et al., 2006). Collectively, these findings illustrate that higher levels of hopelessness were related to and predicted greater levels of depression.

In summary, individuals who demonstrate an increased sense of hopelessness view the future as negative, bleak, and intolerable. Reformulated helplessness and hopelessness theories are specific versions of Beck's cognitive theory, and claim that a pessimistic explanatory style should be correlated with depression symptoms, should be predictive of depressive symptoms over time, and should mediate the relationship between negative cognitions and depression. The hopelessness theory of depression has been well established and evidence has shown that increased levels of hopelessness are associated with greater depression severity. The hopelessness theory of depression, therefore, provides a theoretical framework that outlines the relationship between hopelessness and depression.

Aims and Hypotheses

Dysfunctional beliefs about sleep and hopelessness are likely to be important psychological mechanisms by which insomnia influences depression among older adults. Discovering whether these cognitive variables mediate the insomnia-depression relation will improve the understanding of both sleep and mood problems. Furthermore, exploring a cognitive path model that highlights how insomnia influences depression may better inform future prevention and treatment options for these concerns. No study has yet investigated how insomnia influences depression from a cognitive perspective, or tested a cognitive model that attempts to explain how insomnia predicts depression.

The first aim of the current study was to develop and test a model that explores whether dysfunctional beliefs about sleep and hopelessness mediate the relationship between insomnia and depression among older adults. It was hypothesised that insomnia will predict depression, both directly and indirectly, via dysfunctional beliefs about sleep and hopelessness. The hypothesised path model is depicted in Figure 1.

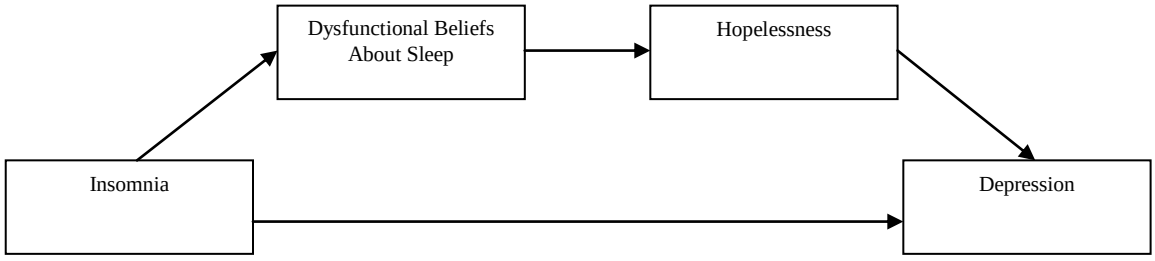

Figure 1. Hypothesised path model.

A second exploratory aim was also investigated in the current study. The majority of older adults with high levels of persistent insomnia have comorbid physiological sleep disorders (Stepanski & Rybarczyk, 2006), such as OSA and/or RLS. These older adults, however, are often not represented within the insomnia-based research because they are excluded for having comorbid sleep problems (Smith et al., 2005; Taylor et al., 2007). Studies have also reported that individuals with physiological sleep disorders exhibit stronger levels of dysfunctional beliefs about sleep compared to individuals without these conditions (Edinger, 2003; Smith et al., 2004). Furthermore, the relationship between insomnia and unhelpful sleep-related beliefs is stronger among people with comorbid sleep conditions compared to people with a single sleep disorder (Smith et al., 2004). This suggests that cognitive factors, such as strongly held sleep-related beliefs, may also play a role in explaining the relationship between insomnia and depression among older adults with physiological sleep disorders. Currently, there are no studies that have investigated whether the paths between insomnia and depression are different among older adults with physiological sleep disorders. Therefore, the present study will test whether the proposed cognitive path model, illustrated in Figure 1, differs according to whether or not participants are likely to have a physiological sleep disorder (OSA and/or RLS).

Method

Participants

The initial sample consisted of 218 Australian older adults aged between 65 and 96 years (M = 74.22, SD = 7.61), including 115 females (53%) and 103 males (47%). Most participants were married (56%), with 44% either being widowed, divorced, de-facto, or single. Fifty six percent had completed secondary school and 44% had completed primary school.

A total of 171 participants completed the follow-up measure of depression, and these participants represented the final sample in the current study. The final sample was also aged between 65 and 96 years (M = 73.94, SD = 7.78), which included 90 females (53%) and 81 males (47%). Most participants were married (58%) and had completed secondary school (56%).

Participants were recruited from retirement villages throughout Victoria, Australia. The term ‘retirement village’ refers to estates built for retired older adults to live independently in their own home. Most people who live in retirement villages are fully functioning individuals who require little assistance with daily living. Since the participants were able to complete the questionnaire and function independently within their own homes, it was assumed that the participants’ cognitive abilities were intact. No exclusion criteria were utilised in the study to increase the chance of obtaining a more accurate representation of the older adult community.

Materials

Each participant received a questionnaire package that consisted of a Plain Language Information Statement, a demographics page, the Insomnia Severity Index (ISI; Morin, 1993), the Dysfunctional Beliefs and Attitudes About Sleep 10-Item Scale (DBAS-10; Espie,

Inglis, Harvey, & Tessier, 2000), the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974), the Snoring Tiredness Observed Pressure Scale (STOP; Chung et al., 2008), and the Restless Legs Syndrome Questionnaire (RLSQ; Allen & Earley, 2001). Participants who were willing to participate in the follow up measure of depression completed the Centre for Epidemiologic Studies Depression Scale (CESD; Radloff, 1977).

Two Plain Language Information Statements (Appendix A) were developed to ensure participants understood the nature of the current study. The first statement outlined pertinent information pertaining to participation in the research project. The second statement outlined pertinent information pertaining to participation in the follow-up measure of depression. For instance, the statements informed participants that each questionnaire would be coded to protect their personal information, and supportive contact details were provided for participants who wanted to discuss their current situation further.

Participants provided demographic information (Appendix B) via a variety of questions. Participants were asked to provide their age, gender, education level, and relationship status. Participants were also asked to indicate whether or not they had been diagnosed with a sleep disorder and/or mental health problem, or were taking medications to assist with sleep or mood. Physical health was measured with two items. First, participants were asked to "Please circle one box to indicate your current level of physical health". The options to this item included 'very poor', 'poor', 'average', 'good', or 'very good'. Second, participants were asked "Do you have any chronic medical conditions?" to which participants circled 'Yes' or 'No'. Finally, participants were asked to circle 'Yes' or 'No' to the following questions, "Would you like to improve you sleep?" and "Would you like to improve your mood?".

The Insomnia Severity Index (Morin, 1993; Appendix C) is a self-report instrument measuring the participant's perception of his or her level of insomnia. The ISI assesses the

subjective symptoms and consequences of insomnia, as well as the degree of concerns or distress caused by those difficulties during the previous two weeks. Its content corresponds in part to the diagnostic criteria of primary insomnia (APA, 2000). The ISI comprises seven items assessing: the severity of sleep-onset and sleep maintenance difficulties (both nocturnal and early morning awakenings); satisfaction with current sleep pattern; interference with daily functioning; ability to notice the level of sleep impairment; and degree of distress or concern caused by the sleep problem. Each item is rated on a five-point scale (0 = none, to 4 = very). Total scores range from 0 to 28, with higher scores representing more severe levels of insomnia. Scores of 0 to 7 indicate no clinically significant insomnia; scores of 8 to 14 indicate sub-threshold insomnia; and scores of 15 to 28 indicate clinical levels of insomnia.

The ISI has been validated in a sample of older adults that underwent treatment for insomnia (Morin et al., 1999). The validity of this instrument was assessed with Pearson's coefficients by correlating insomnia severity with sleep diaries and polysomnography results. Correlations between ISI and sleep diary variables ranged from r = .32 to r = .55 at baseline and from r = .50 to r = .91 at post-treatment. Correlations between ISI and polysomnographic variables ranged from r = .07 to r = .45 at pre-treatment, and r = .23 to r = .45 at post-treatment. The internal consistency of the ISI was estimated by calculating a Cronbach's α coefficient and with an item-total correlation at the pre, post, and follow-up evaluations. Item-total correlations ranged from r = .32 to r = .71, with a mean of r = .56 at pre-treatment; r = .58 to r = .79, with a mean of r = .69 at post-treatment; and r = .46 to r = .90, with a mean of r = .72 at twelve month follow-up. The internal reliability coefficients remained stable from .76 to .78 at follow-up (Morin et al., 1999). The ISI was found to be a reliable measure of insomnia severity in the current sample (Cronbach's α = .91).

The Dysfunctional Beliefs and Attitudes About Sleep 10-Item Scale (Espie et al., 2000; Appendix D) was administered to assess maladaptive thoughts, attitudes, attributions,

and beliefs about sleep (e.g., “I must get eight hours of sleep to feel refreshed and function well during the day”). Participants completed each question using a 10-cm visual analogue scale, anchored with strongly disagree and strongly agree. Added together the 10 item responses provide the final DBAS score. Total scores range from 0-100, with higher scores representing more rigid or stronger levels of dysfunctional beliefs and attitudes about sleep.

The DBAS-10 was redeveloped into 10 items from Morin et al.'s (1993) original 30 item DBAS scale. The 30 item DBAS was originally validated among a sample of older adults with and without insomnia (Morin et al., 1993). Both the DBAS and DBAS-10 have been used with older adults (e.g., Morin et al., 2002). The DBAS-10 has a high validity with Morin et al.'s DBAS scale (r = .83; Espie et al., 2000). Item-total correlations for the DBAS-10 range from r = .45 to r = .73, with the majority being greater than r = .68 (Espie et al., 2000). Psychometric data indicated that the DBAS has excellent internal consistency (Cronbach's α = .80) and an average item-total correlation of r = .37 (Morin et al., 1993). The DBAS-10 was found to have an acceptable Cronbach’s α of .72 (Espie et al., 2000). The DBAS-10 was found to be a reliable measure of dysfunctional beliefs about sleep in the current sample (Cronbach's α = .88).

The Beck Hopelessness Scale (Beck et al., 1974; Appendix E) is a self-report instrument which entails 20 true-false statements designed to assess the degree to which an individual holds negative beliefs about the future over the previous week. Each of the 20 statements is scored 0 or 1 with the total being calculated by summing the pessimistic responses for the 20 items. For example, a participant that answers “True” to the follow question, “My future seems dark to me”, would score 1 point and represent a pessimistic response. The total BHS score ranges from 0 to 20, with higher scores reflecting higher levels of hopelessness. Scores ranging from 0 to 3 identify minimal hopelessness, 4 to 8 identify

mild hopelessness, scores from 9 to 14 identify moderate hopelessness, and scores greater than 14 identify severe hopelessness.

The BHS has been used widely among older adult community samples (e.g., Tanaka, Sakamoto, Ono, Fujihara, & Kitamura, 1996; Walton, Shultz, Beck, & Walls, 1991). Test-retest reliability over a six week period ranges from r = .66 to r = .69, and internal consistency of the BHS ranges from .87 to .93 (Beck & Steer, 1988). The BHS was found to be a reliable measure of hopelessness in the current sample (Cronbach's α = .79).

The Snoring Tiredness Observed Pressure Scale (Chung et al., 2008; Appendix F) is a recently developed self-report tool used to screen for OSA. It contains four yes/no items that helps distinguish between people that are high risk of OSA, and people that are low risk of OSA. Each question relates to a major symptom of OSA. For example, the first item of the measure assesses snoring, “Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?” The second item assesses tiredness, “Do you often feel tired, fatigued, or sleepy during the daytime?” The third item assesses observed apnea episodes, “Has anyone observed you stop breathing during your sleep?” The final item assesses high blood pressure, “Do you have or are you being treated for high blood pressure?” Participants that answer ‘Yes” to two or more items are considered to be at high risk of OSA, and participants that answer ‘Yes’ to less than two items are considered to be at low risk of OSA.

The STOP scale is an effective tool that discriminates well between adults with and without OSA (Chung et al., 2008). Test-retest scores were measured over a 4 week interval, with 96% of participants receiving the same scores upon retesting (Chung et al., 2008). Two independent certified polysomnographic technologists each interpreted the polysomnographic recordings and STOP results in Chung et al.'s study. To avoid bias and inaccuracy from polysomnographic scoring, the polysomnographic recording of 10 randomly selected participants were re-scored by the other experienced technologist, who was blinded to the

score of the other technologist. The scores from the two technologists for the same participants were almost identical (r = .98). Furthermore, Chung et al. reported that the STOP scale was found to be 95% accurate at screening participants that underwent an OSA polysomnogram diagnostic study. The internal consistency of the OSA screening tool was also found be excellent (Cronbach's α = .92, Chung, 2008). The STOP scale was found to be a reliable measure of OSA in the current sample (Cronbach's α = .73).

The Restless Legs Syndrome Questionnaire (Allen & Earley, 2001; Appendix G) is a brief four-item self-report measure of RLS. The RLSQ helps differentiate between participants that are more likely to have RLS, compared to participants who are less likely to RLS. Those who answer "Yes" to all of the following four items are likely to fulfil the diagnostic criteria for RLS as set by the International RLS Study Group (Walters, 1995). The four items are: (1) "I experience a desire to move my legs, which is usually associated by discomfort or disagreeable leg sensations", (2) "I do something to relieve the discomfort from my legs", (3) "The leg discomfort sensations are worse when lying down", and (4) "The leg discomfort sensations are worse later in the day or at night". Participants who answer 'Yes' to all four items are considered to be likely to have RLS, and participants who do not answer 'Yes' to all four items are considered less likely to met the criteria for RLS.

The RLS diagnostic criteria that were set by the International RLS Study Group have been applied in many studies that have investigated RLS, including both adult (e.g., Allen & Earley, 2001; Nichols et al., 2003) and older adult samples (e.g., Cueller, Strumpf, & Ratcliffe, 2007; Rothdach, Trenkwalder, Haberstock, Keil, & Berger, 2000). When the RLSQ was compared with expert clinical diagnoses for consecutive participants who presented to a sleep clinic for objective diagnostic testing, the RLSQ had a sensitivity of 92% and a specificity of 95% (Allen & Earley, 2001). Studies report that the RLSQ has construct validity of .96 and convergent validity between .69 and .90, with an internal consistency of

.93 (Abetz, Arbuckle, & Allen, 2006; Allen & Earley, 2001). The RLSQ was found to be a reliable measure of RLS in the current sample (Cronbach's α = .91).

The Centre for Epidemiologic Studies Depression Scale (Radloff, 1977; Appendix H) is a 20-item self-report questionnaire used to assess depression during the previous week (e.g., "I felt lonely"). Participants responded to each item using a four point scale, where 0 = rarely or none of the time (less than 1 day), to 3 = most or all of the time (5-7 days). When collating the responses, four items are reverse scored. The possible score range was 0 to 60, with higher scores indicating the presence of more depressive symptomatology. Total scores of 16 or above on the CESD indicate the presence of clinically significant levels of depression (Radloff & Teri, 1986).

The CESD is a well-established instrument used to detect depression severity (Radloff, 1977). This measure has been used in many studies among a variety of sample groups, including older adults (Radloff & Teri, 1986). The CESD discriminates well between clinical and community population samples, and highly correlates with similar measures of depression, such as the Beck Depression Inventory (Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977). Test-retest correlations of total scores obtained at 2, 4, 6, and 8 weeks apart averaged r = .57 (Radloff, 1977). Internal consistency of the CESD ranges from .85 to .92 (Radloff & Teri, 1986), with sensitivity of 77% and specificity of 84% (Radloff, 1977). The CESD was found to be a reliable measure of depression in the current sample (Cronbach's α = .90).

Procedure

Approval to conduct the study was granted by the University of Ballarat Human Research Ethics Committee. Face to face recruitment was carried out during December 2010 by attending community group meetings within retirement villages. The questionnaire

packages had been counterbalanced into 20 different versions prior to distribution to control for order effects. Potential participants were invited to complete a self-report questionnaire package. Participants were provided with a Plain Language Information Statement, a questionnaire package, and a reply-paid self-addressed envelope. Once the participants had completed the questionnaire, they were instructed to place the completed questionnaire in the reply-paid self-addressed envelope to post back to the Principal Researcher. Participation in the study was voluntary and informed consent was given by participants reading the Plain Language Information Statement and then indicating their consent to participate by returning the completed questionnaire. At the end of the questionnaire package, participants were asked to provide their name and address if they were willing to complete a measure of depression three months later (Appendix I).

Participants who chose to participate in the follow-up study were mailed out a second Plain Language Information Statement, the CESD, and a reply-paid self-addressed envelope to post back the CESD to the Principal Researcher. To protect the participants' personal identify, a number code was assigned to each questionnaire, allowing for destruction of their personal contact details. The initial questionnaire package took approximately 20 minutes to complete, and the follow-up questionnaire took approximately 5 minutes to complete.

Three hundred questionnaire packages were dispersed during December 2010, of which 218 were returned fully completed, a response rate of 73%. Of those 218 participants that completed the first questionnaire, 171 returned the second follow-up measure of depression in March 2011, a follow-up response rate of 78%. Participants were not reminded to complete the questionnaire and they did not receive any rewards or compensation for participating in the study.

Data Analyses

The data were screened to ensure that statistical analyses could be completed (Francis, 2004; Tabachnick & Fidell, 2001). Only complete data sets were used for analysis, hence there was no missing data. Inter-item correlations were conducted to ensure that no items between measures were multi-collinear (Thompson, 2004). Means and standard deviations were calculated for insomnia, dysfunctional beliefs about sleep, hopelessness, and depression, and correlations between variables were conducted.

The path model was tested using Mplus Version 5.1 (Muthén & Muthén, 2007). The path analysis used the maximum likelihood estimation method with the variance –covariance matrix. Path analyses commonly use Chi-Square (χ^2) as an index of statistical fit. Chi-Square is an index of statistical fit which reflects the closeness of fit between the unconstrained sample covariance matrix and the constrained covariance matrix (the hypothesised model). A significant χ^2 value generally suggests a poor fit. The decision to accept or reject the hypothesised model should not be based purely on statistical grounds, but should consider theoretical and conceptual explanations (Hu & Bentler, 1998).

Practical fit indices were also used to determine the model fit. The practical fit indices used in the current study were the Root Mean Square Error of Approximation (RMSEA), the Comparative Fit Index (CFI), the Tucker – Lewis Index (TFI), and the Standardised Root Mean Square Residual (SRMR). The RMSEA provides a measure of model fit relative to the population covariance matrix where the complexity of the model is also accounted for. RMSEA values less than .06 indicate good fit, values between .06 and .08 indicate a moderate fit, values between .08 and .10 indicate a marginally good fit and values greater than .10 indicate a poor fit (Hu & Bentler, 1998). Hu and Bentler suggest that in sample sizes less than 250, RMSEA and TLI are less preferable fit indices, as is the case in the current study (N = 171). Thus, it is important to consider other practical fit indices to assess the path

model. Hu and Bentler propose that the recommended cut-off value for SRMR is .08, and that values closer to 0.00 indicate a better model fit. The CFI is a measure of the fit of the hypothesised model relative to the independent model, with possible values ranging from 0.00 to 1.00. CFI values in the range of 0.90 to 0.95 indicate an acceptable fit, and values greater than 0.95 indicate good fit (Hu & Bentler, 1998). The TLI, like the CFI, is a measure of comparative fit where values in the range of 0.90 to 0.95 indicate an acceptable fit, and values greater than 0.95 indicate good fit. The guidelines by Hu and Bentler were used in the current study to evaluate the model fit in order to reduce the likelihood of a Type 1 error. As a result, the current study evaluated model fit using χ^2, RMSEA, SRMR, CFI, and TLI values.

The invariance of the path model was explored among participants who were likely and unlikely to have a physiological sleep disorder using the method recommended by Byrne (1998). The method involves computing at least two models with multi-group path analysis. The first of these models, otherwise known as the unconstrained model (Model 1), entails specifying the hypothesised model in all groups so that all hypothesised parameters are free to assume any value. In the second model, otherwise known as the constrained model (Model 2), the path coefficients for the hypothesised model for one of the two groups are specified so that the parameters are free to assume any value; while in the second group, these parameters are constrained to be identical to the first model.

The test for group invariance entails a comparison of the χ^2 values across the models. A non-significant difference in χ^2 suggests group invariance for both the measurement and structural models. A significant difference in χ^2 value suggests group non-invariance, meaning the models are similar despite differing groups. One approach that can be used to find the non-invariant parameters is to examine the modification indices, along with the associated expected parameter change statistics. Large modification indices are identified,

and the path involving this parameter is released rather than constrained. The χ^2 value of this revised model is then compared to the original model (Model 1). A non-significant difference in χ^2 suggests that all but the freely estimated parameter are invariant across the groups. If the difference between the χ^2 values of Model 1 and the model with the freed path remains significant, the modification indices are examined once again and the path with the next largest modification index is freed. If required, this process can be continued until all non-invariant parameters are identified, as indicated by a non-significant difference in χ^2 values. Once a non-significant difference in χ^2 values has been reached, this indicates that all paths that have been freely estimated are invariant across the groups.

Results

Data obtained from the questionnaire packages were entered using SPSS for Windows Version 17. The path model was tested using Mplus Version 5.1.

Additional Demographic Information

A longitudinal design was used to investigate how insomnia, dysfunctional beliefs about sleep, and hopelessness related to depression over the period of three months. Two hundred and eighteen participants completed the initial questionnaire package. Of those participants, 171 completed the follow-up measure of depression. Participants who completed the follow-up measure of depression were considered to be the final sample.

Demographic information of the final sample demonstrated that the majority of participants reported having good (35%) or average physical health (35%); 13% reported having very good health, 13% reported having poor health, and 4% reported having very poor health. Approximately half of the sample reported having at least one chronic medical condition (48%). Eighteen percent reported that they had been diagnosed with a sleep

disorder, and 15% reported that had been diagnosed with a mental health problem. Further demographic results indicated that over half of the sample (60%) wanted to improve their sleep, and 39% of participants wanted to improve their mood. Almost one-quarter (23%) of participants scored at or above the clinical cut-off score for insomnia, and 19% scored at or above the clinical cut-off score for depression.

Comparison Between Completers and Non-Completers

Of the initial sample that completed the questionnaire package (N = 218), forty seven participants did not complete the follow-up measure of depression, which left a final sample of 171 participants.

An Independent Samples *t*-Test was conducted to investigate whether the mean age was different between participants who completed the follow-up measure of depression and participants who did not complete the follow-up measure of depression. It was found that the mean age was not significantly different between each group, t (216) = -1.05, $p > .05$.

Table 1 illustrates demographic information pertaining to participants who completed the follow-up measure of depression and participants who did not complete the follow-up measure of depression. Chi-Square tests indicated that there were no significant relationships between group and demographic variables, as shown in Table 1.

Table 1

Demographic Information for Participants who Completed the Follow-Up Measure of Depression and for Participants who did not Complete the Follow-Up Measure of Depression

Demographics	Completers (N = 171)		Non-Completers (N = 47)		
	n	%	*n*	%	χ^2
Gender					
Male	81	47	22	47	0.23
Female	90	53	25	53	0.46
Relation Status					
Married	100	58	26	55	3.03
Not Married	71	42	21	45	2.48
Education Level					
Primary	75	44	21	45	1.24
Secondary	96	56	26	55	1.81
Medication					
Yes	67	40	20	43	4.20
No	104	60	27	57	4.43
Medical Condition					
Yes	82	48	22	47	0.15
No	89	52	25	53	0.36

Note. * Indicates χ^2 difference significant at $p < .05$.

The means, standard deviations, skew and kurtosis results for measures of participants who completed the follow-up measure of depression and for participants who did not complete the follow-up measure of depression are shown in Table 2. The skew and kurtosis values ranged from -0.05 to 1.09 and -1.34 to 1.11, respectively. Although these results suggested that the distribution of the data departed slightly from normality, it has been suggested that this only becomes problematic when skew values approach 2.0 and kurtosis

7.0 (Curran, West, & Finch, 1996). Hence, the skew and kurtosis values were within reasonable bounds.

An Independent Samples *t*-Test was also conducted to investigate whether the mean scores on measures were different between participants who completed the follow-up measure of depression and for participants who did not complete the follow-up measure of depression. As shown in Table 2, mean scores on measures did not significantly differ for participants who completed the follow-up measure of depression compared to participants who did not complete the follow-up measure of depression.

Table 2

Descriptive Information of Measures for Participants who Completed the Follow-Up Measure of Depression and for Participants who did not Complete the Follow-Up Measure of Depression

	Completers (n = 171)				Non-Completers (n = 47)				
Measure	*M*	*SD*	Skew	Kurtosis	*M*	*SD*	Skew	Kurtosis	*t*
ISI	8.65	6.39	0.55	-0.75	9.23	6.19	0.05	-1.34	-0.55
DBAS	43.16	20.48	0.02	-0.58	44.49	18.24	-0.05	-0.75	-0.40
BHS	4.88	3.64	1.09	1.11	5.74	3.58	0.69	0.75	-1.44
CESD	10.94	9.67	1.01	0.47	-	-	-	-	-

Note. ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; BHS = Beck Hopelessness Scale; CESD = The Centre for Epidemiologic Studies Depression Scale.
*$p < .05$.

Testing the Model

Correlation analyses were conducted to investigate the relationships between variables. Table 3 shows the correlation matrix for measures of participants who completed

the follow-up measure of depression. Table 4 shows the covariance matrix for the observed measures of participants who completed the follow-up measure of depression.

Table 3

Correlation Matrix of the Final Sample (N = 171)

Measure	1	2	3	4
1. ISI	1.00			
2. DBAS	.61**	1.00		
3. BHS	.50**	.49**	1.00	
4. CESD	.65**	.56**	.62**	1.00

Note. ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; BHS = Beck Hopelessness Scale; CESD = The Centre for Epidemiologic Studies Depression Scale.
***p* < .01.

Table 4

Covariance Matrix of the Final Sample (N =171)

Measure	1	2	3	4
1. ISI	40.91			
2. DBAS	79.15	419.55		
3. BHS	11.72	36.86	13.24	
4. CESD	40.58	111.13	21.78	93.56

Note. ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; BHS = Beck Hopelessness Scale; CESD = The Centre for Epidemiologic Studies Depression Scale.

Path Analysis

The first aim of the current study was to develop and test a model to explore whether dysfunctional beliefs about sleep and hopelessness mediate the relationship between insomnia and depression among older adults. It was hypothesised that insomnia would predict

depression, both directly and indirectly, via dysfunctional beliefs about sleep and hopelessness. For participants who completed the follow-up measure of depression, fit indices for the hypothesised path model, as specified in Figure 1, were as follows: χ^2 (2) = 20.97, $p < .01$, CFI = .93, TLI = .79, SRMR = .07, RMSEA = .23, RMSEA 90% CI = .15 - .33. The CFI and SRMR indices indicated an acceptable fit, however, the χ^2, TLI, and RMSEA indices indicated a poor fit. Overall, fit indices for the hypothesised model indicated that the model was a poor fit.

Path analysis allows testing for modification indices. One modification was added to the model in an attempt to improve the fit. This involved adding a direct path from insomnia to hopelessness. The fit indices of the revised path model, as depicted in Figure 2, were as follows: χ^2 (1) = 5.35, $p = .02$, CFI = .98, TLI = .91, SRMR = .02, RMSEA = .16, RMSEA 90% CI = .05 -.30. All five fit indices indicated that the revised path model was a very good fit. No further modification indices were recommended and this model was accepted as the final model. Figure 2 demonstrates the standardised path estimates for the revised path model.

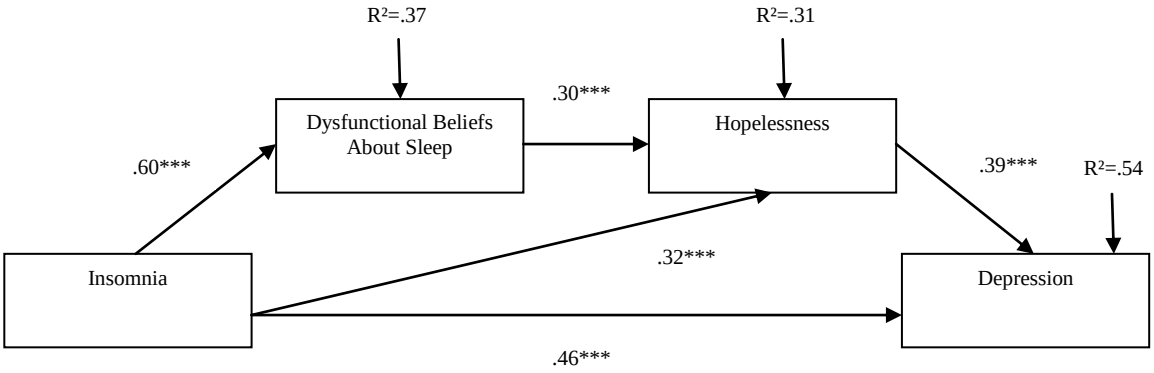

Figure 2. Revised path model.
***$p < .001$.

Path analysis allows examination of both the direct and indirect effects within the model (Baron & Kenny, 1986). Direct effects are represented by single straight arrows from one variable to another. As shown in Figure 2, all paths within the model are significant. Mediation effects are also demonstrated in the model. Full mediation indicates that the mediators explain all of the relationship between the predictor and dependent variables. Partial mediation occurs when the direct effects of the predictor on the dependent variable are diminished but not eradicated by the intervening or mediator variables. The predictor, therefore, maintains a relationship with the dependent variable, even after controlling for the mediators (Baron & Kenny, 1986). As depicted in Figure 2, there is a significant, direct effect of insomnia on depression.

Path analysis allows the examination of the total indirect effects for the model, that is, the impact of insomnia on depression via dysfunctional beliefs about sleep and hopelessness. Results indicated that there was partial mediation for the model as the indirect path from insomnia to depression via dysfunctional beliefs about sleep and hopelessness was also significant, $r = .09$, $p < .01$. In addition, the indirect path from insomnia to depression via hopelessness was also significant, $r = .13$, $p < .01$. Therefore, the revised path model represents a partially mediated model.

Testing the Revised Path Model for Invariance

A second exploratory aim was investigated in the present study. This involved testing whether the revised path model, as depicted in Figure 2, differed according to whether or not participants were likely to have a physiological sleep disorder (OSA and/or RLS). Participants were divided into two groups based on the scoring criteria suggested by the STOP (Chung et al., 2008) and RLSQ (Allen & Earley, 2001) scales. Of the 171 participants that completed the follow-up measure of depression, 87 were likely or at high risk of having a

physiological sleep disorder (OSA and/or RLS), and 84 were unlikely or at low risk of having a physiological sleep disorder.

The means, standard deviations, skew and kurtosis results for measures of participants who were likely and unlikely to have a physiological sleep disorder who completed the follow-up depression measure are shown in Table 5. The skew and kurtosis values ranged from -0.40 to 1.60 and -0.29 to 3.26, respectively. The skew and kurtosis values for this analysis were within reasonable bounds (Curran et al., 1996).

An Independent Samples t-Test was conducted to investigate whether the mean scores on measures differed depending on the likelihood of participants having a physiological sleep disorder (OSA and/or RLS). As shown in Table 5, the mean scores of each measure was significantly higher for participants who were likely to have a physiological sleep disorder compared to participants who were unlikely to have a physiological sleep disorder.

Table 5

Descriptive Information for Participants who were Likely and Unlikely to Have a Physiological Sleep Disorder on All Measures

	Likely Sleep Disorder (n = 87)				Unlikely Sleep Disorder (n = 84)				
Measure	*M*	*SD*	Skew	Kurtosis	*M*	*SD*	Skew	Kurtosis	*t*
ISI	10.78	6.41	0.14	-0.83	6.35	5.56	1.15	0.49	4.80**
DBAS	50.57	20.12	-0.40	-0.35	35.11	17.74	0.28	0.19	5.31**
BHS	6.02	3.89	0.88	0.59	3.65	2.89	1.29	1.95	4.50**
CESD	14.33	9.85	0.73	-0.29	7.26	8.03	1.60	3.26	5.13**

Note. ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; BHS = Beck Hopelessness Scale; CESD = The Centre for Epidemiologic Studies Depression Scale.
**$p < .01$.

Table 6 shows the test for invariance between the likely physiological sleep disorder group and the unlikely physiological sleep disorder group. As shown in Table 6, there was no significant difference between the χ^2 values for the two groups when all paths were constrained to be equal. This indicates that the model was non-invariant for the physiological sleep disorder and no physiological sleep disorder group.

Table 6

Tests for Invariance Between the Likely Physiological Sleep Disorder Group and the Unlikely Physiological Sleep Disorder Group

Model	χ^2	*Df*	Model comparison	$\Delta\chi^2$	Δdf
Baseline Disorder and No Disorder free (A)	3.95	2			
Disorder and No Disorder equal (B)	5.93	7	A vs B	1.98	5

Note. * Indicates χ^2 difference significant at $p < .05$.

In summary, results indicated that the model fit for participants who completed the follow-up study was generally poor. One modification that included a direct path from insomnia to hopelessness substantially improved the model fit. Participants who were likely to have a physiological sleep disorder (OSA and/or RLS) reported higher scores on all four measures compared to participants who were unlikely to have a physiological sleep disorder. Results further indicated that there was no significant difference in model fit between the physiological sleep disorder and no physiological sleep disorder group. Consequently, the revised path model was accepted as the final model for all participants, regardless of whether or not they were likely to have a physiological sleep disorder.

Discussion

The primary purpose of the current study was to develop and test a model that explored whether dysfunctional beliefs about sleep and hopelessness mediated the relationship between insomnia and depression among older adults. It was hypothesised that insomnia would predict depression, both directly and indirectly, via dysfunctional beliefs about sleep and hopelessness. The results of the first path analysis indicated an overall poor model fit.

Consequently, a revised model was tested, which included one additional direct path from insomnia to hopelessness. Results indicated an excellent model fit, with all paths in the model being significant. The revised path model was considered a partial mediation model, as the relationship between insomnia and depression remained significant after the inclusion of the mediating variables.

A second exploratory aim investigated whether the revised path model differed according to whether or not participants were likely to have a physiological sleep disorder (OSA and/or RLS). Results indicated that there was no significant difference in model fit between the physiological sleep disorder and no physiological sleep disorder groups. The revised path model was therefore accepted as the final model for all participants, regardless of whether or not they were likely to have a physiological sleep disorder.

Applicability of the Revised Path Model

Epidemiological studies have reported that insomnia is one of the most prevalent problems that affect older adults (Foley et al., 2004; Neubauer, 2005; Smith et al., 2005). Furthermore, research has shown that older adults are twice as likely to suffer with severe levels of insomnia compared with younger adults (Doghramji, 2006).With a rapidly ageing population worldwide (WHO, 2006), the incidence of insomnia and depression is likely to

increase, which will place greater demand on the healthcare system (Aminzadeh et al., 2002; Crimmins, 2004; Qualls et al., 2002).

There is a growing body of literature that has investigated the relationship between insomnia and depression. Substantial evidence exists to support the suggestion that high levels of insomnia increase the risk of developing increased depression severity (e.g., Buysse et al., 2008; Pigeon & Perlis, 2007; Riemann & Voderholzer, 2003; Taylor et al., 2005). Fewer studies, however, have investigated the relationship between insomnia and depression among older adults. Consistent with previous research (e.g., Perlis et al., 2006), the revised path model indicated that there was a direct relationship between insomnia and depression, even after the inclusion of maladaptive sleep beliefs and hopelessness. This provides further evidence that suggests insomnia severity is strongly and directly related to depression levels among older adults.

While the current study has supported previous research by reporting the relationship between insomnia and depression, no previous study has attempted to explain how insomnia influences depression from a cognitive perspective. The present study drew on theoretical and empirical research to construct a model that proposed a cognitive pathway from insomnia to depression. The revised path model can be understood with reference to previous research that has demonstrated the importance of psychological mechanisms related to insomnia and depression.

Cognitive conceptualisations of insomnia suggest that rigidly held sleep-related beliefs play a critical role in maintaining insomnia (Carney & Edinger, 2006; Ellis et al., 2007; Harvey, 2002; Morin et al, 1993; Morin et al., 2007). Consistent with these studies, the final model indicated a direct association between insomnia and maladaptive sleep beliefs. As a result, an older individual who reported higher levels of insomnia was likely to endorse stronger problematic sleep-related beliefs (e.g., “I can’t sleep! I won’t be able to function this

week if I can't get to sleep"). This provides further evidence that older adults' with higher levels of insomnia experience unhelpful cognitions that are not conducive to productive sleep.

The revised path model also provides support to Harvey's (2002, 2005) cognitive model of insomnia. Harvey's model highlighted how cognitive mechanisms, such as dysfunctional beliefs about sleep, function to perpetuate insomnia. Harvey proposed that insomnia was maintained by several interrelating cognitive processes that operate at night and during the day. One of the central aspects to Harvey's cognitive model of insomnia that was supported by the current study involved the endorsement of dysfunctional beliefs about sleep.

Harvey's (2002) model proposed that dysfunctional beliefs about sleep were closely interrelated with several cognitive processes (e.g., negatively toned cognitive activity, arousal/distress, selective attention, misperception), which serve to exacerbate these unhelpful cognitive mechanisms. For example, an individual who is experiencing persistent negatively toned cognitive activity during the pre-sleep period is likely to develop a strongly held belief that they need to stay in bed and try harder to fall asleep. This means that the unhelpful cognitive mechanisms which occur during the pre-sleep period increase the intensity of the maladaptive sleep-related cognitions.

Harvey (2002) proposed that the relationship between insomnia and dysfunctional beliefs could also occur at various times throughout the night, causing disruptions to sleep maintenance. This is particularly relevant to older adults because this proportion of the population often awake more during the night (Shochat et al., 2007; Vitiello, 2006). For instance, an older adult with greater levels of insomnia could wake up during the night to go to the bathroom and upon returning to bed, endorse unhelpful beliefs making return to sleep difficult. Harvey's model postulates that the rigid endorsement of dysfunctional beliefs about

sleep contributes further to psychological and physiological distress during the night, consolidating the interrelated maladaptive cognitive cycle that perpetuates insomnia.

In summary, the final model presented in the current study highlighted the strong relationship between insomnia and depression among older adults. This provided support to an increasing body of research that had suggested high levels of insomnia increases the risk of developing depressive symptoms among older adults (Mallon et al., 2000; Perlis et al., 2006; Riemann & Voderholzer, 2003). In addition, previous research has established that older adults with persistent insomnia problems report more intense dysfunctional beliefs about sleep compared to older adults with low levels of insomnia, a finding that became one of the foundations to Harvey's (2002) cognitive model of insomnia. As expected, the path between insomnia and dysfunctional beliefs about sleep in the final model indicated that higher levels of insomnia directly predict more rigid maladaptive sleep cognitions among older adults.

The present study identified another important, yet, unexplored psychological construct within the insomnia literature that could play a significant role in further understanding how insomnia and dysfunctional beliefs about sleep influence depression. This involved the psychological construct of hopelessness. There are no previous studies that have empirically tested whether hopelessness plays a role in the relationship between insomnia and depression. The relationship between unhelpful sleep beliefs and hopelessness, however, was briefly commented on in Morin et al.'s (1993) influential study. Morin and colleagues noticed that older adults with insomnia endorsed stronger beliefs about the negative consequences of insomnia, expressed more hopelessness about the fear of losing control of their sleep, and experienced more helplessness about its unpredictability compared to older adults without insomnia. Until now, no study had collectively investigated the paths between between insomnia, dysfunctional beliefs about sleep, hopelessness, and depression.

The current research was the first study to predict that maladaptive sleep beliefs and hopelessness would play a role in understanding how insomnia related to depression. As expected, an indirect path was found between insomnia and depression via maladaptive sleep beliefs and hopelessness. Consistent with Morin et al.'s (1993) proposal, older adults who reported higher levels of insomnia endorsed more intense inflexible sleep-related cognitions, which in turn, predicted an increased sense of hopelessness and then, depression. Although no previous research has attempted to explain the relationship between these variables, Harvey's (2002, 2005) cognitive conceptualisation of insomnia may help understand how individuals who report high levels of insomnia could develop an increased sense of hopelessness via strongly held dysfunctional beliefs about sleep.

Harvey's (2002, 2005) cognitive model of insomnia suggested that daytime cognitive processes are assumed to be of equal importance to the maladaptive processes that operate at night. This pattern of night and day dysfunctional cognitive processes is where an underlying sense of hopelessness could develop. Given that people with insomnia tend to automatically underestimate the amount of sleep they obtain and usually engage in the appraisal process of sleep when they wake in the morning (Clark, 1999; Harvey, 2004; Tang & Harvey, 2004), it is likely that the person will go into the day with the belief that they did not get enough sleep. These thoughts will be particularly intense if the perceived sleep debt has been accumulating over a long period of time (Harvey, 2004). Consequently, it is likely that a person with persistent insomnia will be preoccupied with unhelpful sleep-related cognitions, such as being preoccupied with fatigue and tiredness, not coping, and not performing adequately during the day. Several additional daytime beliefs are also likely to occur for an individual with insomnia, such as concerns about losing control of their ability to sleep and falling ill as a result of inadequate sleep, which in turn could increase a sense of loss of control and negativity toward the future. This pervasive negative bias during the day and night provides

an example of how persistent insomnia and strongly held dysfunctional beliefs about sleep could influence an individual's sense of hopelessness.

While Harvey's (2002) cognitive model of insomnia did not explicitly discuss the concept of hopelessness, several other elements within Harvey's model demonstrate how hopelessness could result from high levels of insomnia and dysfunctional beliefs about sleep. For example, Harvey suggested that individuals with insomnia who selectively attend to and intensely monitor their environment are likely to increase the chance of detecting ambiguous cues (e.g., feelings of tiredness in the legs) that can then be misinterpreted (e.g., "I mustn't have slept enough last night"), further influencing the misperception of other daytime events (e.g., "I'm not coping"). The unhelpful interpretation of seemingly minor events could occur numerous times during the day and night. These negative experiences are likely to build up over time because the dysfunctional beliefs remain unchanged or disproven, which could possibly contribute towards an increased sense of hopelessness. Consequently, suffering from high levels of insomnia and endorsing rigid maladaptive sleep-related beliefs could consolidate a pessimistic view about the future, as sleep continues to be disturbed. These examples from Harvey's model illustrate how a sense of hopelessness could develop among people with high levels of insomnia and dysfunctional beliefs about sleep.

It was expected that dysfunctional beliefs about sleep and hopelessness would be central to understanding how insomnia is related to depression. To further highlight the importance of these psychological mechanisms, two additional indirect paths were also supported by the revised model. The first of these two additional indirect paths involved the relationship between dysfunctional beliefs about sleep, hopelessness, and depression. As expected, older adults who endorsed stronger levels of unhelpful sleep-related beliefs were more likely to report higher levels of hopelessness, which in turn, predicted greater depression severity.

The additional indirect path presented within the revised model was an unpredicted, but theoretically sound relationship between insomnia, hopelessness, and depression. Specifically, it was found that the relationship between insomnia and depression was partially mediated by hopelessness. These indirect paths collectively indicated that hopelessness, in addition to dysfunctional beliefs about sleep, was an important cognitive element to consider when understanding the relationship between insomnia and depression among older adults. Since Harvey's cognitive model of insomnia was heavily influenced by Beck's theory of depression (e.g., Beck, 1963, 1976; Beck et al., 1979), reflecting upon Beck's research can assist to further understand how dysfunctional beliefs and hopelessness influenced the relationship between insomnia and depression in the current study.

Cognitive models, such as Harvey's (2002) model of insomnia and Beck's (1976) model of depression, share the premise that maladaptive thinking and negative appraisals of daily events contribute to the development and perpetuation of insomnia and depression. Beck's cognitive model postulates that three psychological concepts explain the development of depression, which includes the cognitive triad, schemas, and cognitive errors. Each of these theoretical concepts can be applied to the proposed path model, which demonstrates the importance of dysfunctional beliefs and hopelessness in explaining the insomnia-depression relation.

One aspect of Beck's (1976) cognitive triad was particularly relevant to the current study, which contained the theme of hopelessness. This element involves an individual anticipating that their current difficulties will continue indefinitely. For instance, an individual may make long-term projections based on their current circumstances, expecting that there will be unremitting hardship, frustration, and despair. As a result, when the individual considers undertaking a specific task in the immediate future, they expect to fail (Beck, 1976). This component of Beck's cognitive triad could be applied to the model

proposed in the current study. An older adult who consistently experiences problems with insomnia may strongly believe that their sleep will never improve, and in turn, a sense of hopelessness could develop. The hopelessness element of Beck's cognitive triad, therefore, provides a useful theoretical understanding of how individuals with high levels of insomnia and dysfunctional beliefs about sleep could experience an increased sense of hopelessness.

The revised path model also lends support to the second component of Beck's (1976) model, which consisted of the concept of schemas. Beck proposed that when a person is confronted with a particular situation, a schema related to the circumstance is activated. Beck stated that schemas constituted an automatic cognitive response that moulded neutral pieces of information into thoughts or beliefs that develop into emotions. Beck proposed that an individual tends to develop consistencies in their responses to similar types of events. For example, an individual who continues to find it difficult to initiate sleep could develop negative schemas that are activated during the pre-sleep period. Consequently, relatively stable cognitive patterns form the basis for the regularity of interpretations of a particular set of situations (e.g., attempting to fall asleep), which could develop increased levels of hopelessness due to schemas becoming activated in sleep-related situations.

The third component to Beck's (1976) theory of depression that can be applied to the current model is cognitive errors. Beck proposed that those individuals at risk of developing depression distorted reality in a systematic manner that resulted in a bias against the self, their environment, and the future. Beck outlined several cognitive errors that demonstrate faulty information processing. For example, absolutistic thinking (otherwise known as black/white thinking), refers to a tendency to place all experiences in one of two opposite categories (e.g., good verses bad). Cognitive errors are inherent within dysfunctional beliefs about sleep. For instance, an individual who experiences consecutive nights of poor sleep may develop a strongly held belief that "I'm a bad sleeper", demonstrating an example of an absolutistic

cognitive error. These cognitive processing errors can influence overly negative and pessimistic interpretations, evaluations, and appraisals of the current and future context. Furthermore, Beck observed that people at risk of depression tended to view their experience as total defeats (non-dimensional) with irreversible consequences (fixed). These individuals tended to categorise themself as a 'loser' (judgemental) and doomed (irreversible character deficits). The emotions connected with these thoughts, thus, were likely to be negative and possess a heightened sense of hopelessness and depression.

In summary, the revised path model indicated that insomnia and dysfunctional beliefs about sleep were both important predictors of hopelessness, indicating that older adults who reported increased levels of insomnia were likely to express more intense dysfunctional beliefs about sleep, and in turn, report a greater sense of hopelessness. Beck's (1976) and Harvey's (2002) cognitive models were useful frameworks to explain how dysfunctional beliefs and hopelessness partially mediated the relationship between insomnia and depression. This collection of theoretical concepts and empirical findings suggest that psychological constructs, such as dysfunctional beliefs about sleep and hopelessness, play important roles in understanding how insomnia predicts depression among older adults.

The revised path model provides further support for Abramson et al.'s (1989) hopelessness theory of depression. As was previously discussed in Beck's (1963, 1976) theory of depression, individuals who demonstrate an increased sense of hopelessness perceive the future as negative, bleak, and intolerable. Reformulated helplessness and hopelessness theories (e.g., Abramson et al., 1978) were specific versions of Beck's cognitive theory, and claimed that a pessimistic explanatory style should be correlated with depressive symptoms, should be predictive of depressive symptoms over time, and should mediate the relationship between dysfunctional beliefs and depression.

The hopelessness theory of depression (Abramson et al., 1989) is still an accepted theory that suggests hopelessness predicts depression severity. This theory proposed that individuals with a pessimistic explanatory style tend to attribute the causes of negative events to global and stable factors, whereas individuals with an optimistic explanatory style exhibit the tendency to attribute such causes to specific and unstable factors (Abramson et al., 1989). It was suggested that individuals with a pessimistic explanatory style are also more likely to make depressive inferences about the causes of negative events than individuals without this style. Such consistent inferences increase the likelihood of hopelessness, and once hopelessness develops, depression is inevitable, since hopelessness is viewed as a proximal and sufficient cause of depression in this theory (Abramson et al., 1989).

The hopelessness theory of depression (Abramson et al., 1989) has been well established and evidence has shown that increased levels of hopelessness predict greater depression severity (e.g., Alloy et al., 2006; Isaacowitz & Seligman, 2001). Consistent with these studies, the revised path model illustrated that insomnia and inflexible sleep beliefs were related to depression via hopelessness. Specifically, older adults with high levels of insomnia and dysfunctional beliefs about sleep were likely to report greater levels of depression via an increased sense of hopelessness. As a result, the revised path model provided further support to the hopelessness theory of depression (Abramson et al., 1989).

Overall, the revised path model presented in the current study demonstrated significant indirect and direct effects of insomnia on depression. The model indicated that there was a direct relationship between insomnia and depression. The model also suggested that in addition to this direct relationship, the relationship between insomnia and depression was partially mediated by dysfunctional beliefs about sleep and hopelessness. This finding was consistent with theoretical and empirical research that proposed relationships between

insomnia, maladaptive sleep beliefs, hopelessness, and depression (e.g., Abramson et al., 1989; Beck, 1976; Harvey, 2002; Morin et al., 1993).

Test for Differences Between Groups

A second exploratory aim was also investigated in the present study. This involved testing whether the revised path model differed according to whether or not participants were likely to have a physiological sleep disorder (OSA and/or RLS). It appears that only two previous studies (Edinger, 2003; Smith et al., 2004) have investigated physiological sleep conditions from a psychological perspective. Both of these studies indicated that individuals with physiological sleep disorders endorsed high levels of problematic sleep-related cognitions. Consistent with Edinger (2003) and Smith et al.'s (2004) research, the current study found that participants who were likely to have a physiological sleep disorder reported significantly higher levels of dysfunctional beliefs about sleep compared to participants who were unlikely to have a physiological sleep disorder. In addition, the current study found that participants who were likely to have a physiological sleep disorder also reported higher scores on four all key measures (e.g., insomnia, dysfunctional beliefs about sleep, hopelessness, depression) compared to participants who were unlikely to have a physiological sleep disorder. This suggests that older adults who were likely to have a physiological sleep disorder were at greater risk of experiencing higher levels of insomnia, dysfunctional beliefs about sleep, hopelessness, and depression compared to older adults who were unlikely to have a physiological sleep disorder. Although these results add to a limited body of research, the second exploratory aim in the current study endeavoured to test whether the revised path model differed according to whether or not participants were likely to have a physiological sleep disorder.

No previous study has tested whether a cognitive pathway from insomnia to depression differed depending on an older adult's physiological sleep disorder status. Results indicated that there was no significant difference in model fit between the likely physiological sleep disorder group and unlikely physiological sleep disorder group, indicating that the revised path model was applicable to all participants, regardless of whether or not they were likely to have a physiological sleep disorder. Moreover, the revised path model indicated that dysfunctional beliefs about sleep and hopelessness helped explain how insomnia influenced depression for all participants. This result provides evidence that cognitive mechanisms, such as dysfunctional beliefs about sleep and hopelessness, are equally applicable to individuals who are likely and unlikely to have a physiological sleep disorder.

Since the model was found to be non-invariant between the physiological sleep disorder group and no physiological sleep disorder group, important academic and clinical implications emerge for the sleep field. This finding supports the notion that participants with comorbid sleep conditions (e.g., insomnia and OSA) should be included in research that investigates the assessment and treatment of sleep disorders. Currently, most studies (e.g., Edinger et al., 2008; Edinger & Means, 2005; Morin et al., 1999; Sivertsen et al., 2006; Wang et al., 2005) that investigate insomnia have excluded participants with physiological sleep disorders. The present findings, however, suggest that the cognitive pathway between insomnia and depression was similar for individuals who were likely to have a physiological sleep disorder. This provides further evidence that cognitive mechanisms may play a role in further understanding physiological sleep disorders.

Future research that integrates both medical and psychological perspectives of physiological sleep problems is clearly warranted. Combining this knowledge should not only improve the understanding of sleep disorders, but could ultimately improve treatment options

for individuals who experience comorbid sleep conditions (e.g., insomnia and OSA). The results from the current study have several more important implications for both professionals who work with older adults and for older adults who reside within the community.

Community Implications

Previous research has suggested that insomnia is one of the most misunderstood and undetected problems among all health conditions (Benca, 2005; Bootzin & Epstein, 2011; Doghramji, 2006; Morin, 2004). The revised path model, therefore, could assist in enhancing the awareness of insomnia and its potential consequences (e.g., depression) among older adults. Since general practitioners are usually the central health provider that older adults regularly attend (Aminzadeh et al., 2002; Qualls et al., 2002), educating general practitioners about the cognitive pathway from insomnia to depression could play a vital role in better detecting and understanding the occurrence of insomnia within the community.

Distributing the results of the present study to general practitioners could improve their awareness of insomnia and depression among older adults. This enhanced recognition and understanding could translate into general practitioners conducting more vigorous insomnia assessments with their patients. If persistent insomnia problems are evident, for instance, general practitioners could become more aware that a referral to a specialist service may be required (e.g., clinical psychologist, sleep physician), since these older adults present a greater risk of developing higher levels of depression via unhelpful sleep beliefs and hopelessness.

To assist senior citizens who require further sleep assessment, government bodies or private organisations could consider subsidising these specialist sleep consultations. This financial benefit may provide an extra incentive for older adults to follow through with getting their sleep assessed. This first step of obtaining a thorough sleep assessment and

receiving appropriate treatment could possibly prevent future problems for older adults, such as depression.

There are additional opportunities that could be considered within the community to promote a better understanding and awareness of sleep among older adults. For example, developing media campaigns that are directed towards older adults and older adult carers could help these individuals become more aware of their sleep pattern and promote regular sleep check-ups with their general practitioner. Sleep-focussed agencies or groups could also be established within communities to encourage this growing proportion of the population seek further advice and support about potential sleep problems. Implementing these strategies could play a major role in highlighting the importance of sleep health among the older adult community. It is likely that improving community awareness will save money and resources, as correctly detecting and treating sleep disorders could prevent future illnesses (Bootzin & Epstein, 2011; Taylor et al., 2003).

Clinical Implications

In addition to community-based implications, the current research provides valuable information for clinicians who work with older adults. For instance, the revised path model is useful for conceptualising how insomnia influences depression among older adults. This information could form the basis for psychological formulations to assist both healthcare providers and older adults better understand the relationship between insomnia and depression. Specifically, the revised path model indicated that older adults who experience higher levels of insomnia are at an increased risk of developing depression via strongly held dysfunctional beliefs about sleep and an increased sense of hopelessness. Consequently, collaboratively addressing each element within the cognitive model could help clients build insight into how insomnia is impacting their mood. The proposed model could therefore

provide a platform for developing idiosyncratic psychological conceptualisations with clients who are presenting with high levels of insomnia.

Support for the revised path model indicates that insomnia, dysfunctional beliefs about sleep, and hopelessness are important elements to consider when developing prevention and/or treatment strategies for depression among older adults. The results obtained in the current study provide evidence that cognitive-based therapies could be important treatment options for addressing insomnia and depression among older adults. Cognitive therapy, for instance, is an evidence-based, structured, time-limited approach that grew from Beck and colleagues' (1979) research (DiCaccavo, 2010; Freeman, Pretzer, Fleming, & Simon, 2004; McCurry et al., 2007). Cognitive therapy for depression highlighted the close relationship between negative cognitive processes, hopelessness, and depression (Beck et al., 1979). Beck proposed that altering negative thoughts with cognitive reframing techniques would improve hopelessness and depression. For instance, an individual who automatically thinks “I’m not getting enough sleep, so I won’t be able to function this week”, is encouraged to systematically evaluate the belief, and practice reframing it into a more balanced response, such as “I’ve been able to function before with little sleep, so it might turn out to be a good week”. This exercise is designed to help individual’s become flexible in their thinking and shift their attention towards more helpful cognitions.

More recently, Harvey (2005) used a cognitive model to develop a cognitive therapy program for insomnia. The aim of this treatment program was to correct the unhelpful cognitive processes that served to maintain insomnia. The core treatment components to this line of therapy involved formulation, psycho-education, cognitive restructuring, behavioural experiments, and relapse prevention. The application of these cognitive-based interventions was collectively designed to reduce levels of insomnia in the long term by constructing more

positive maintenance cycles (Bootzin & Epstein, 2011; Harvey, 2005; McCurry et al., 2007; Morin, 2004).

While Harvey's (2005) cognitive therapy program for insomnia provides a base for future research, little empirical evidence is available to validate this treatment, particularly among older adults. In addition, no research has yet endeavoured to test whether treating insomnia with cognitive therapy could improve and /or prevent depression among older adults. Since the relationship between insomnia and depression was partially mediated by dysfunctional beliefs about sleep and hopelessness in the current study, insomnia-based cognitive interventions that aim to improve the flexibility of sleep-related beliefs and enhance levels of hopefulness will likely have a positive effect on reducing depression severity. It is therefore suggested that future studies explore how treating insomnia with cognitive interventions impact depression levels among older adults.

Studies that have evaluated the treatment of insomnia have failed to specifically address how improving hope could impact treatment outcomes. There are some studies, however, that have discussed the role of improving hope in a clinical setting with older adults (e.g., Bergin & Walsh, 2005; Farran, Herth, & Popovitch, 1995; Karel & Hinrichsen, 2000). Farran et al. proposed that hope involves the ability to be flexible and adaptive in striving for a desired object or outcome. Moreover, Karel and Hinrichsen suggested that one of the most consistent themes, in effective psychotherapy, is inspiring hope within the client that change is possible. As such, the experience of being hopeful, as opposed to hopeless, elicits the stamina required to effect real change (Bergin & Walsh, 2005; Karel & Hinrichsen, 2000). More specifically, the client's hopes are believed to be located within specific achievable goals that are made accessible by the client's perception that they are working within a realistic formulation and treatment (Bergin & Walsh, 2005; Farran et al., 1995). Indeed, what separates hope from wishful thinking or blind optimism is the realistic achievability of the

hoped-for outcome (Bergin & Walsh, 2005). Thus, part of the hope engendered by therapy arises from working towards manageable, achievable, and valued psychological goals that are collaboratively developed within the therapeutic program (Bergin & Walsh, 2005).

In summary, the revised path model presented in the current study highlights the important role of dysfunctional beliefs and hopelessness in influencing the relationship between insomnia and depression. Specifically, an older adult who experiences persistent high levels of insomnia and dysfunctional sleep beliefs is at risk of developing depression via greater levels of hopelessness. It is possible that future insomnia treatment programs that also aim to improve an older adult's level of hopelessness could reduce and/or potentially prevent depression symptoms.

Although the current study has provided insight into a preliminary path model that offers a cognitive explanation of how insomnia relates to depression among older adults, the results should be viewed in light of some limitations.

Limitations

Several limitations exist within the current study that merit consideration for future research. The first limitation of the present study relates to the use of path analysis to analyse the data. While path analysis is accepted as being a robust statistical technique, it only allows for the examination of relationships between observed variables. Understanding of the proposed model may be enhanced via structural equation modelling, which allows the examination of relationships between latent constructs and reduces potential measurement errors by having multiple measures of each latent variable. It must also be remembered that it is not possible to infer causal relationships between variables with a path analysis design.

When investigating the relationships between variables, collecting data over a period of time is preferable to better test the direction of the relationships. Although the current

study collected data at two time points separated by a three month interval, this was still a relatively short longitudinal design. Due to restrictions in the project's timeline, it was not possible to conduct a more comprehensive longitudinal study. It is suggested that future studies collect data over a six to twelve month period, as this would further strengthen the directional component of the cognitive path model.

Although longitudinal studies are preferred over studies that collect and analyse data concurrently, longitudinal research is often limited by participant 'drop-out' rates. Despite the convenience of attracting an initial reasonable sample size (N = 218), 22% of the original sample did not complete in the follow-up measure of depression. Therefore, the current study was only able to analyse the data of 171 participants who completed the follow-up measure of depression. Although it was not expected that all participants would complete the follow-up measure of depression, a drop-out rate of 22% was still important to recognise, especially in a relatively short longitudinal study. This limitation was neutralised as statistical analyses showed no significant differences between participants who completed the follow-up measure of depression and participants who did not complete the follow-up measure of depression. It is suggested that future studies aim to collect a more comprehensive sample size, which would further strengthen the generalisability of the results. In addition, future studies could offer participants incentives/rewards (e.g., shopping vouchers) to maximise the chance of retaining participants over time.

Another limitation that was inherent within the current study related to the applicability of the findings to other populations. For instance, as the study used a community sample, the extent to which the findings could be generalised to clinical groups is uncertain. Furthermore, the study intentionally focussed specifically on older adults, so the interpretability of the results to individuals under the age of 65 years is also unclear. Future research may wish to investigate how the results of the current study differ between clinical

and community groups, or between adults and older adults. Collecting information from a community sample, however, was particularly important in this study because many older adults in the community experience sleep and mood problems that often remain undetected (Benca, 2005; Doghramji, 2006; Smith et al., 2005).

The current study was also limited by reliance on self-report measures. As a result, the findings may have been confounded by common method variance. To reduce the impact of relying solely on self-report data collection, the current study counterbalanced the questionnaire packages to improve the random distribution of measures to participants. Ideally, future studies should validate the results obtained here by collecting data from multiple methods (e.g., self-report, diagnostic interview, sleep diary) and from different sources (e.g., partner, next of kin).

A similar data collection limitation was evident in the study's second exploratory aim, which involved the assessment of physiological sleep disorders. Self-report measures were used to differentiate participants into two groups based on their likelihood of having a physiological sleep disorder. Since sleep disorders such as OSA and RLS have a physiological basis, it is recommended that these conditions be assessed by a sleep technician team via a polysomnogram diagnostic study, which requires access to a clinical sleep laboratory and medical practitioner (Smith et al., 2004). Recently, it has been suggested that researchers who do not have access to a sleep laboratory could use an actigraph watch (Vallieres & Morin, 2003), which is designed to collect objective data to assist physiological sleep assessment. Despite these 'gold-standard' measures of physiological sleep disorders, the budget and timeframe restrictions of many research projects do not allow the use of these expensive, time-intensive assessment methods. Consequently, the accuracy of participant allocation in the current study was limited by reliance of self-report. To counteract this limitation, the study used reliable self-report measures that had been validated among various

populations. In addition, the present study ensured that the two groups were described using terms such as ‘likely’ and ‘unlikely’ to have a physiological sleep disorder, instead of using more definitive diagnostic expressions when describing the two groups. Despite this, the ability of the current study to generalise to those with a formally diagnosed physiological sleep disorder remains limited.

Notwithstanding these limitations, the results indicated an excellent model fit and represented a new way of understanding how insomnia influences depression among older adults. Moreover, this study offers clear evidence that the revised model is applicable to older adults that reside within the community, whether they are likely to have a physiological sleep disorder or not, and presents important areas for intervention and future research.

Future Research and Conclusion

The current study is the first of its kind to offer a cognitive explanation of how insomnia influences depression among older adults. The revised path model indicated significant indirect and direct effects of insomnia on depression, indicating that this was a partially mediated model. Consistent with previous research, the model indicated that there was a direct relationship between insomnia and depression. The partial mediation effect demonstrated in the model suggests that in addition to the direct relationship, the relationship between insomnia and depression was partially mediated by dysfunctional beliefs about sleep and hopelessness. This finding was consistent with theoretical and empirical research that proposed relationships between insomnia, maladaptive sleep beliefs, hopelessness, and depression. It was therefore concluded that dysfunctional beliefs about sleep and hopelessness helped explain how insomnia influenced depression among older adults.

The results from the second exploratory aim suggested that the revised path model was non-invariant between groups, indicating that the model was applicable to all participants

regardless of whether or not they were likely or to have a physiological sleep disorder. The revised path model was particularly relevant to older adults who were likely to have a physiological sleep disorder, as these participants reported significantly higher levels of insomnia, dysfunctional beliefs about sleep, hopelessness, and depression compared to participants who were unlikely to have a physiological sleep disorder. These findings suggest that future research should consider including participants with comorbid sleep conditions (e.g., insomnia and OSA). In addition, it is suggested that the exploration of cognitive variables (e.g., maladaptive sleep beliefs) in future studies may improve the understanding of how physiological sleep disorders are maintained.

The revised path model provides a framework that future research can build from to continue improving the understanding of how insomnia influences depression. It is important to recognise that the current study tested a preliminary model that only explored two mediating variables (dysfunctional beliefs about sleep and hopelessness). Although the proposed path model may be limited by its preliminary nature, 54% of the variance in depression was explained by the model. This illustrates that an older adult's level of insomnia, dysfunctional beliefs about sleep, and hopelessness are important factors to consider when conceptualising depression severity.

While the model proposed in the present study offers a preliminary cognitive pathway from insomnia to depression via dysfunctional beliefs and hopelessness, it does not explain all the variance in depression. This suggests that the inclusion of additional variables in future studies could enhance the understanding of how insomnia influences depression. There are some supplementary factors that could extend the understanding of the proposed path model. For instance, people with insomnia often undertake in certain behaviours (e.g., napping, alcohol use, withdrawing from activities) that unintentionally exacerbate their sleep problems (Bootzin & Epstein, 2011; Harvey, 2002; McCurry et al., 2007; Ree & Harvey, 2004). As a

result, including a behavioural element to the cognitive model may provide more information about how insomnia relates to depression. In addition, older adults are more likely to experience more physical health problems compared to younger adults (Crimmins, 2004), and studies have reported that increased levels of insomnia are related to poorer health (Taylor et al., 2003; Taylor et al., 2007). Consequently, incorporating a physical health component could make the model more specific to an older adult population.

The foundations of the revised path model offer important implications for the future treatment of insomnia and depression among older adults. Future research is required to investigate whether cognitive-based treatment programs that target insomnia, dysfunctional beliefs, and hopelessness improve and/or prevent depression among older adults. Overall, educating both healthcare professionals and community-dwelling older adults about the cognitive pathway from insomnia to depression could enable more efficient recognition, treatment, and prevention of these highly prevalent conditions.

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Appendix A – Plain Language Information Statement

Plain Language Information Statement

University of Ballarat
Learn to succeed

SCHOOL OF BEHAVIOUR, SOCIAL SCIENCE, & HUMANITIES

PROJECT TITLE:	**"Sleep and mood among older Australians"**
PRINCIPAL RESEARCHER:	**Associate Professor Suzanne McLaren**
OTHERS / STUDENT RESEARCHERS:	**Dr Megan Jenkins & Mr Paul Sadler**

Dear Potential Participant,

We sincerely thank you for your interest in this study being conducted by Mr Paul Sadler, a Masters by Research candidate, alongside Associate Professor Suzanne McLaren and Dr Megan Jenkins from the University of Ballarat. This study will investigate how sleep influences mood among older adults. The information that is being collected will be of value to health professionals seeking to assist older adults with potential sleep and mood problems.

If you volunteer to participate in this research, you will be asked to complete a questionnaire, which asks for some background information, and various questions about your sleep and mood. The attached questionnaire will take approximately 20-30 minutes to complete. It is very important that you answer each question as truthfully as possible for the research to be of significance. You will also have the option of completing a brief questionnaire, assessing mood, three months after completing the whole questionnaire package. This is valuable, as it will allow us to assess how your sleep is related to your mood over time. This is explained in detail on the last page of the questionnaire. Please be assured that completing this initial questionnaire is of value, even if you do not wish to do the follow up questionnaire in three months time.

If you want to participate, please use the accompanying "reply paid" envelope to return the completed questionnaire to the researchers. Please note that returning the questionnaire is an indication that you understand the nature of the research and that you are freely volunteering to participate in the research. When your questionnaire is completed and returned, they will form a larger database from which only group data will be reported. Your individual results will not be reported and none of the information that you supply in this study will be able to be traced or linked back to you in any way. The information you provide will be strictly confidential by allocating a code to your completed questionnaire, which will de-identify your personal details to protect your identity. You may withdraw your participation from this research at any time during the completion of the questionnaire (particularly if you are experiencing distress). However, please understand that once you have returned the completed questionnaire, it will be impossible to identify your responses among the larger pool and therefore withdrawal at this stage will not be an option. Questionnaires will be kept for a period of five years after any publications that arise from this study.

You are encouraged to discuss any questions that you may have during, or at the conclusion of the questionnaire, with the Principal Researcher Associate Professor Suzanne McLaren or your doctor. Should you prefer to discuss your issues anonymously, you may wish to contact Lifeline (available 24 hours a day: telephone 13 11 14 for the cost of a local call or free call 1300 651 251). Please contact Associate Professor Suzanne McLaren to obtain a copy of the results, which will be available in August 2011.

If you have any questions, or you would like further information regarding the project titled **(Sleep and mood among older Australians)**, please contact the Principal Researcher, **(Suzanne McLaren)** of the School of **(Behaviour, Social Science, & Humanities)**: **PH**: (03) 5327 9628, **EMAIL:** s.mclaren@ballarat.edu.au

Should you (i.e. the participant) have any concerns about the ethical conduct of this research project, please contact the University of Ballarat Ethics Officer, Research & Graduates Studies Office, University of Ballarat, PO Box 663, Mt Helen VIC 3353. Telephone: (03) 5327 9765, Email: ub.ethics@ballarat.edu.au; CRICOS Provider Number 00103D

Appendix A – Plain Language Information Statement

Plain Language Information Statement

University of Ballarat
Learn to succeed

SCHOOL OF BEHAVIOUR, SOCIAL SCIENCE, & HUMANITIES

PROJECT TITLE:	**"Sleep and mood among older Australians"**
PRINCIPAL RESEARCHER:	**Associate Professor Suzanne McLaren**
OTHER/STUDENT RESEARCHERS:	**Dr Megan Jenkins & Mr Paul Sadler**

Dear Participant,

Thank you for completing our questionnaire approximately three months ago. At that time, you indicated a willingness to receive a short follow-up questionnaire. If you want to complete this questionnaire, please continue. If you have changed your mind, that is OK, and we thank you for completing our first questionnaire. We assure you that the first questionnaire is still of value to us, even if you choose not to complete this final questionnaire.

If you would like to complete this follow up questionnaire, please do so over the next few days and return it to the Principal Researcher via the enclosed reply paid self-addressed envelope. The information you provide will be strictly confidential by allocating a code to your completed questionnaire, which will de-identify your personal details to protect your identity.
You are encouraged to discuss any questions that you may have during, or at the conclusion of the questionnaire, with the Principal Researcher Associate Professor Suzanne McLaren or your doctor. Should you prefer to discuss your issues anonymously, you may wish to contact Lifeline (available 24 hours a day: telephone 13 11 14 for the cost of a local call or free call 1300 651 251). If you wish, please contact Associate Professor McLaren to obtain a copy of the results, which will be available August 2011.

We sincerely thank you again for participating in this research.

If you have any questions, or you would like further information regarding the project titled **(Sleep and mood among older Australians)**, please contact the Principal Researcher, **(Suzanne McLaren)** of the School of **(Behaviour, Social Science, & Humanities)**: **PH**: (03) 5327 9628, **EMAIL:** s.mclaren@ballarat.edu.au
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Appendix B – Demographic Information

1. Age _____ (yrs) **2.** Please circle: Female† Male†

3. Please circle your current relationship status:

Married† Divorced† Widowed† Single† De-facto†

4. Please circle the highest level of education you have achieved:

Primary Secondary Tertiary

5. Have you been diagnosed with a sleep disorder?

No† Yes†

6. Have you been diagnosed with a mental health problem?

No† Yes†

7. Do you currently take any medications to assist your sleep or mood?

No Yes

8. Would you like to improve your sleep?

No† Yes†

9. Would you like to improve your mood?

No† Yes†

10. Please circle your current level of physical health compared to others your age:

Very Poor† Poor† Average† Good† Very good†

11. Do you have any chronic medical conditions?

No† Yes†

Appendix C – Insomnia Severity Index

Instructions:

Please circle the response that best resembles you for each question. Please answer all questions.

1. Please rate the current (last two weeks) severity of your insomnia problem(s):

	None	**Mild**	**Moderate**	**Severe**	**Very**
Difficulty falling asleep:	0	1	2	3	4
Difficulty staying asleep:	0	1	2	3	4
Problem waking up too early:	0	1	2	3	4

2. How satisfied/dissatisfied are you with your current sleep pattern?

Very Satisfied	Satisfied	Neutral	Dissatisfied	Very Dissatisfied
0	1	2	3	4

3. To what extent do you consider your current sleep problem to interfere with your daily functioning (e.g. daytime fatigue, ability to function at work/daily chores, concentration, memory, mood, etc.).

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering
0	1	2	3	4

4. How noticeable to others do you think your sleeping problem is in terms of impairing the quality of your life?

Not at all Noticeable	Barely	Somewhat	Much	Very Much Noticeable
0	1	2	3	4

5. How worried/distressed are you about your current sleep problem?

Not at all	A Little	Somewhat	Much	Very Much
0	1	2	3	4

Appendix D – Dysfunctional Beliefs and Attitudes About Sleep 10-Item Scale

Instructions:

Several statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement, circle the number that corresponds to your own personal belief. Please respond to all items even though some may not apply directly to your situation.

1. I need 8 hours sleep to feel refreshed and function well during the day.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

2. When I don't get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

3. I am concerned that chronic insomnia may have serious consequences on my physical health.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

4. When I have trouble getting to sleep, I should stay in bed and try harder.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

5. I am worried that I may lose control over my abilities to sleep.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

6. After a poor night's sleep, I know that it will interfere with my daily activities on the next day.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

7. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

8. When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

9. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

10. I get overwhelmed by my thoughts at night and often feel I have no control over this racing mind.

Strongly Disagree_____Strongly Agree
0 1 2 3 4 5 6 7 8 9 10

Appendix E – Beck Hopelessness Scale

Instructions:

This questionnaire consists of 20 statements. Please read the statements carefully one by one. If the statement describes your attitude for **the past week including today**, circle the " T " indicating TRUE in the column next to the statement. If the statement does not describe your attitude, circle the " F " indicating FALSE in the column next to this statement. **Please be sure to read each statement carefully.**

1	I look forward to the future with hope and enthusiasm	T	F
2	I might as well give up because there is nothing I can do about making things better for myself	T	F
3	When things are going badly, I am helped by knowing that they cannot stay that way forever	T	F
4	I can't imagine what life would be like in ten years	T	F
5	I have enough time to accomplish the things I want to do	T	F
6	In the future, I expect to succeed in what concerns me most	T	F
7	My future seems dark to me	T	F
8	I happen to be particularly lucky, and I expect to get more of the good things in life than the average person	T	F
9	I just can't get the breaks and there's no reason I will in the future	T	F
10	My past experiences have prepared me well for the future	T	F
11	All I can see ahead of me is unpleasantness rather than pleasantness	T	F
12	I don't expect to get what I really want	T	F
13	When I look ahead to the future, I expect I will be happier than I am now	T	F
14	Things just won't work out the way I want them to	T	F
15	I have great faith in the future	T	F
16	I never get what I want, so it's foolish to want anything	T	F
17	It's very unlikely that I will get any real satisfaction in the future	T	F
18	The future seems vague and uncertain to me	T	F
19	I can look forward to more good times than bad times	T	F
20	There's no use in really trying to get anything I want because I probably won't get it	T	F

Appendix F - Snoring Tiredness Observed Pressure Scale

Instructions:

Please circle Yes **"Y "** or No **" N "** to the following questions that are related to your sleep behaviours.

1	Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?	Y	N
2	Do you often feel tired, fatigued, or sleepy during daytime?	Y	N
3	Has anyone observed you stop breathing during your sleep?	Y	N
4	Do you have or are you being treated for high blood pressure?	Y	N

Appendix G - Restless Legs Syndrome Questionnaire

Instructions:

Please circle Yes **"Y "** or No **" N "** to the following questions that are related to your sleep behaviours.

1	I experience a desire to move my legs, which is usually associated by discomfort or disagreeable leg sensations.	Y	N
2	I do something to relieve the discomfort from my legs.	Y	N
3	The leg discomfort sensations are worse when lying down.	Y	N
4	The leg discomfort sensations are worse later in the day or at night.	Y	N

Appendix H – Centre for Epidemiologic Studies Depression Scale

Instructions:

Below is a list of some of the ways you may have felt or behaved. Please indicate how often you have felt this way **during the past week** by circling the appropriate number. Please answer all questions.

	Rarely or none of the time (less than 1 day)	Some of a little of the time (1-2 days)	Occasionally or moderate amount of the time (3-4 days)	Most or all of the time (5-7 days)
1. I was bothered by things that usually don't bother me	0	1	2	3
2. I did not feel like eating; my appetite was poor	0	1	2	3
3. I felt that I could not shake off the blues even with help from my family or friends	0	1	2	3
4. I felt that I was just as good as other people	0	1	2	3
5. I had trouble keeping my mind on what I was doing	0	1	2	3
6. I felt depressed	0	1	2	3
7. I felt that everything I did was an effort	0	1	2	3
8. I felt hopeful about the future	0	1	2	3
9. I thought my life had been a failure.	0	1	2	3
10. I felt fearful	0	1	2	3
11. My sleep was restless	0	1	2	3
12. I was happy	0	1	2	3
13. I talked less than usual	0	1	2	3
14. I felt lonely	0	1	2	3
15. People were unfriendly	0	1	2	3
16. I enjoyed life	0	1	2	3
17. I had crying spells	0	1	2	3
18. I felt sad	0	1	2	3
19. I felt that people disliked me	0	1	2	3
20. I could not get 'going'	0	1	2	3

Appendix I – Follow-Up Questionnaire Invitation

Follow-Up Questionnaire

University of Ballarat
Learn to succeed

Please Read Below:

In order to help us understand more about how sleep is related to mood in the long term, we are inviting participants to complete the 20-item measure of mood again in 3 months time. If you complete the second questionnaire, we will need to match it to this one, and that requires you giving us some identifying information. For example, if you write your postal address on this questionnaire, you will be asked to write your postal address on the follow up questionnaire in 3 months time. The two questionnaires can then be matched to explore how sleep is linked with mood.

If you would like to assist further with this project, you will need to write your home or postal address and name on this sheet of paper. In order to protect your identity, we will assign a code to this questionnaire, remove this sheet of paper with your details on it from this questionnaire, and destroy your personal details. We will then write your code on the second questionnaire and address the envelope, ready to post the second questionnaire to you in 3 months time.

This sheet of paper with your contact details on it will be destroyed, protecting your identity.

Please be assured that completing this initial questionnaire is of significant value, even if you do not want to do the follow up questionnaire in 3 months time.

*** I am interested in doing the 20-item measure of mood again in 3 months time.

My name and postal address is _____

_____

_____

_____

Please Note: Even if you choose to receive the follow up questionnaire, you are free to decide NOT to complete it in 3 months time. Should you choose not to do the follow up questionnaire, we will not contact you. Your contact details will have been destroyed.